In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 16-989V
(To be Published)

K.A.,	*	
	*	
Petitioner,	*	
	*	Chief Special Master Corcoran
v.	*	
	*	Dated: April 18, 2022
	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
	*	
Respondent.	*	
	*	

Robert J. Krakow, Law Office of Robert Krakow, P.C., New York, NY, for Petitioner.

Nina Ren, U.S. Dep't of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On August 11, 2016, K.A. filed a petition seeking compensation under the National Vaccine Injury Compensation Program ("Vaccine Program").[2] Petitioner alleges that he experienced Guillain Barré syndrome ("GBS") due to the administration of a Tetanus Diphtheria acellular-Pertussis ("Tdap") vaccine on August 12, 2013. Petition at 1 (ECF No. 1).

[1] This Decision will be posted on the Court of Federal Claims's website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012)). **This means that the Decision will be available to anyone with access to the internet.** As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the Decision's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public. *Id*.

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) ("Vaccine Act" or "the Act"). Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

I proposed (after the case's transfer to me in January 2021) that the matter could reasonably be decided on the record, and the parties have offered briefs in support of their respective positions. Petitioner's Motion, dated July 2, 2021 (ECF No. 84) ("Mot."); Respondent's Opposition, dated Sept. 13, 2021 (ECF No. 88) ("Opp."); Petitioner's Reply, dated Oct. 12, 2021 (ECF No. 89) ("Reply"). Now, after review of the medical record, briefs, and multiple expert reports, I deny entitlement. Petitioner has not preponderantly established that the Tdap vaccine can cause GBS, under either of the two causation theories ventured over the case's six-year life—and even if he had, the record demonstrates it is far more likely his GBS was attributable to an intercurrent upper respiratory infection ("URI") than vaccination.

I. Factual Background

Vaccination and Onset of Neurologic Issues/Symptoms

Mr. K.A., a medical researcher, was fifty-one years old when he received a Tdap vaccine on August 12, 2013, through his employer. Ex. 1 at 2. The medical evidence filed in this case pertaining to Petitioner's pre-vaccination history suggests he suffered from high cholesterol, hypertension, and a chronic leg condition for which he underwent surgery several years post-vaccination. *See* Ex. 2 at 2; Ex. 18; Ex. 75 at 57, 111. Petitioner acknowledges that "[w]ithin a short time" after receiving the vaccine, he experienced a "bad headache, body pains, sore throat and fever," which he believed to be "a routine flu." Ex. 10 at 8. Thus, he was experienced flu-like symptoms prior to the onset of his alleged vaccine-caused neurologic injury, and these symptoms appear broader than the common post-vaccination malaise.

Less than three weeks later, on September 1, 2013, Petitioner went to the North Shore University Hospital ("North Shore") Emergency Department ("ED"), where he reported three days (meaning beginning on or around August 29th) of flu-like symptoms (dry cough, chills) and a feeling of swelling in his throat, which he had been attempting to self-treat with over-the-counter medications based on the supposition that he had some kind of URI. Ex. 9 at 6, 9, 17. On exam, ED treaters noted that Petitioner appeared neurologically sound and was not in distress, although they observed him to display many common URI symptoms (fever, chills, weakness, nasal discharge, congestion, dyspnea, cough, etc.). *Id.* at 11, 17. Mr. K.A. was ultimately discharged from North Shore the next day, and he maintains he was still experiencing a fever on September 3, 2013. *Id*. at 6–11; Ex. 10 at 5.

Then, on September 4, 2013, Petitioner was discovered on his driveway complaining of numbness on his left side, and transported by ambulance to the St. Francis Hospital ED in Roslyn, New York. Ex. 3 at 8–9. Emergency responders noted that he was displaying an unsteady gait, and also that he had recently been treated for flu-like symptoms. Ex. 2 at 5–6. Upon arrival at the hospital, Mr. K.A. informed treaters that his neurologic symptoms had begun "approximately" five days ago,

"when he was attempting to climb a hill and noticed weakness in the left leg," followed by a feeling of "incoordination" on the left side of his face while brushing his teeth. Ex. 3 at 9. (Petitioner contradictorily represented in this same record, however, that "[t]he current episode" began seven days before (*Id.*)). He had not experienced any loss of sensation, but felt tingling generally over the left side of his body. *Id*. Overall, Petitioner deemed his symptoms "mild to moderate." *Id*.

Evaluation and Treatment

On exam, admitting physician Subhash Viswanathan, M.D., noted that Mr. K.A. could not forcefully close his left eye, and that he had left face weakness plus left arm and leg weakness. Ex. 3 at 10–12. His cranial nerves appeared intact, however, and he displayed no sensory defect. *Id*. at 12. CT scans of Petitioner's brain and chest were unremarkable, although lab results showed a positive IgG for West Nile virus ("WNV"), but a negative IgM.[3] *Id.* at 15–20, 63. A lumbar puncture supported the diagnosis of GBS of the "pharyngeal-cervical-brachial variant." *Id.* at 67. A neurologist who saw Petitioner at this time, Michael Han, M.D., concurred in the proposed GBS diagnosis, taking specific note of the fact that Petitioner had recently experienced a URI. Ex. 3 at 15, 19–20.

Mr. K.A. was subsequently admitted to the hospital for further evaluation. Ex. 3 at 16. Weakness was his initial primary complaint, and in providing a history Petitioner noted that he had experienced a URI with flu-like symptoms, thereafter, developing the left leg weakness that had resulted in his ED visit on September 4th. *Id.* at 20, 21. An MRI of the brain was performed after Petitioner's admission but revealed no acute changes. *Id.* at 30. Another neurologist, Teresa Deangelis, M.D., examined Petitioner the next day (September 5th), noting again the URI preceding his neurologic symptoms, and she began Petitioner on a five-day course of intravenous immunoglobulin therapy.[4] *Id.* at 36–37. Petitioner also underwent a rheumatology consult with William Given, M.D., on September 6, 2013, and Dr. Given opined (consistent with the other treaters who had by this time seen Petitioner) that "[t]he patient is . . . a gentleman who has developed weakness and paresthesias following what appears to be a viral illness a couple of weeks ago." *Id.* at 41.

Petitioner also had at this time an infectious disease consult with Dava Klirsfeld, M.D., providing the same history he had given other treaters (URI, subsequent symptoms, etc.)—although this time he included the fact that he had received the Tdap vaccine three weeks prior as well. Ex. 3 at 47–

[3] As I noted in a prior decision, "Immunoglobulin G (IgG) and Immunoglobulin M (IgM) are antibodies produced in response to infection, and their titer levels can help monitor or detect immune deficiencies. IgM is an indicator of current infection, while IgG reflects exposure to a past infection." *Knorr v. Sec'y of Health & Hum. Servs*., No. 15-1169V, 2018 WL 6991548, at *4 n.7 (Fed. Cl. Spec. Mstr. Dec. 7, 2018).

[4] Intravenous immunoglobulin, or "IVIG," is a blood product used to treat patients with antibody deficiencies, including neurological disorders. Clinical Uses of Intravenous Immunoglobulin, NCBI (2005), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1809480/ (lasted visited on Apr. 13, 2022). It is commonly prescribed to treat diseases believed to be autoimmune in nature, with the aim of increasing the effectiveness of an individual's immune response.

48. Dr. Klirsfeld's assessment included the view that Petitioner had experienced "a demyelinating disorder possibly *post viral or post vaccine*." *Id*. at 53 (emphasis added). Dr. Klirsfeld also deemed the positive WNV IgG serum test "of unclear significance," and ordered more laboratory testing. *Id*.

A second lumbar puncture was consistent with the prior one, and on September 10, 2013, Doina Glodan, M.D., diagnosed petitioner with "Guillain-Barré syndrome post recent viral URI." Ex. 3 at 126, 217. Other treaters who saw Petitioner in this time frame also seemed to accept the URI as likely causal. *See,* e.g., *id.* at 127 (Joyce Ott, PT, recording that Petitioner had a history of "recent viral URI"), 130 (Dr. Deangelis noting "recent URI 2 weeks ago"), 154 (David Brieff, M.D., opining that "[p]ositive WNV serology likely reflects old infection or cross reaction from other flavivirus infection in past"). But Petitioner did not test positive for any specific infections, such as the Epstein-Barr virus ("EBV"). *Id.* at 268–85.

Mr. K.A. remained at St. Francis Hospital until September 17, 2013, after which he was discharged to a rehabilitation facility. Ex. 3 at 213, 216. His coordination had now improved, although he continued to report facial weakness, fatigue, and back and shoulder pain. *Id.* There, he attended physical, occupational, and speech therapy, remaining in rehab until September 22, 2013. Ex. 12 at 8. On that date, however, Petitioner developed acute right facial weakness with hemiparesis, and was accordingly transferred to North Shore on the same day for treatment and evaluation. *Id.* He now displayed increasing lethargy, weakness, and right sided numbness and tingling that started two to three days prior, with trace reflexes and worsened right-side issues (in contrast to improvements on his left side). Ex. 9 at 44–49; Ex. 5 at 27.

Petitioner was admitted to North Shore based on his emerging "right side numbness/tingling weakness" in the setting of "recently diagnosed AIDP,"[5] and treaters began him on a course of plasmapheresis[6] on September 24, 2013. Ex. 5 at 30; Ex. 9 at 48. This treatment was continued for some time, and Petitioner's symptoms improved as a result. Ex. 9 at 53. Additional testing performed during this hospitalization revealed a recent cytomegalovirus ("CMV") infection, but was equivocal regarding the presence of EBV. Ex. 9 at 178–80. Notably, on September 26, 2013, a dietician who saw Petitioner indicated in the record from that visit that Petitioner had initially presented "with progressive weakness post tetanus shot 8 weeks ago," although it appears from this record that the dietician was merely taking down a history rather than offering an informed view on causation. *Id*. at 1064.

[5] AIDP refers to "acute inflammatory demyelinating polyradiculoneuropathy" *Polyradiculoneuropathy*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=40276 (last visited Apr. 13, 2022).

[6] Plasmapheresis is the process of drawing blood, removing the plasma therefrom, and replacing it with another substance, such as albumin or type-specific fresh frozen plasma. *Plasmapheresis*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=39455&searchterm=plasmapheresis (last visited Apr. 13, 2022). It is often employed in the treatment of disease processes believed to be autoimmune in nature.

Mr. K.A. was discharged for further rehabilitation at a different facility on October 8, 2013, and he underwent rehab therapy through October 21st before being released for additional outpatient therapy. Ex. 9 at 57; Ex. 7 at 78; Ex. 12 at 193–540. In the course of that treatment, he informed caregivers of his view that his symptoms followed (and hence were potentially related to) his receipt of the Tdap vaccine. Ex. 7 at 5–7. For the remainder of that fall, Petitioner saw Dr. Han for follow-up treatment, and Dr. Han noted Petitioner's overall improvement. Ex. 5 at 58–60, 62.

Treatment in 2014 and Beyond

There is a subsequent, almost nine-month gap in the medical record, with Petitioner not visiting Dr. Han again until September 2014. Ex. 5 at 65. Petitioner then reported that he still was experiencing painful paresthesias in his feet, but no longer needed to use a cane despite some imbalance issues, and described himself as "99% back to normal." *Id*. Dr. Han advised Mr. K.A. that he should continue on his existing medication, and asked him to return in four to six months. *Id*. at 67. Petitioner received a comparable positive evaluation from a different treater. Ex. 75 at 69, 130. Similar prognoses were offered in 2015. *See,* e.g., *id.* at 68, 126 (2015 visits with Dr. Han); Ex. 5 at 80–81 (2016 visits with Dr. Han). No other records from the subsequent timeframe bear on the causation issues presented in this case.

II. Expert Reports

A. Lawrence Steinman, M.D.

Dr. Steinman submitted five expert reports on behalf of Petitioner. Report, dated Aug. 2, 2017, filed as Ex. 21 (ECF No. 30-1) ("First Steinman Rep."); Report, dated Feb. 18, 2018, filed as Ex. 22 (ECF No. 40-1) ("Second Steinman Rep."); Report, dated Dec. 27, 2018, filed as Exhibit 46 (ECF No. 54-1) ("Third Steinman Rep."); Report, dated Oct. 7, 2019, filed as Exhibit 48 (ECF No. 61-1) ("Fourth Steinman Rep."); Report, dated June 22, 2020, filed as Exhibit 71 (ECF No. 74-1)("Fifth Steinman Rep."). Altogether, Dr. Steinman offered more than 70 pages of expert opinion on this matter, (as discussed below and throughout this Decision), I do not conclude the significant effort was ultimately well spent.

As shown in his CV, Dr. Steinman received his B.A. from Dartmouth College and his M.D. from Harvard Medical School. Ex. 24 at 1 (ECF No. 48-2) (Dr. Steinman's Curriculum Vitae ("Steinman CV")). He then completed residencies in neurology and pediatrics at Stanford University. Steinman CV at 1. He has worked as a professor of neurology and pediatrics at Stanford for the past forty-one years (thirty-seven years at the time of filing). *Id.* Dr. Steinman has also published over four hundred peer-reviewed publications on immunology, neurology, and autoimmune disease. *Id*. at 5–45. He has special expertise in the study of immunology, having several articles published on the issues. *Id.* at 5–45. Dr. Steinman is part of the American

Association of Immunologists and the Clinical Immunology Society, with patents in the field and many papers on the topic. *Id.* at 2.

Dr. Steinman has demonstrated expertise in treatment of GBS and associated neuroinflammatory conditions, and has been honored in his field of practice for his specific expertise in the study of multiple sclerosis. First Steinman Rep. at 2–3. And he is a frequent expert in the Vaccine Program. By his own count (as of 2017, meaning over four years ago), Dr. Steinman had testified 20 times—although (as a somewhat recent decision establishes) by the spring of 2019 his reports and opinions had been featured in 40 published Vaccine Program decisions. *D.G. v. Sec'y of Health & Hum. Servs*., No. 11-577V, 2019 WL 2511769, at *191 n.171 (Fed. Cl. Spec. Mstr. May 24, 2019).

First Report

Dr. Steinman began his report with an overview of GBS, which he characterized as an inflammatory neuropathy singling out peripheral nerves and that was likely autoimmune in pathogenesis. First Steinman Rep. at 4–6. He also noted that the record strongly supported the appropriateness of the diagnosis in this case, adding that Respondent did not appear to disagree with it. *Id.* at 6. In addition, there was some record evidence of potential alternative explanations (spider bites; presence of WNV antibodies in CSF testing; Petitioner's URI), and Dr. Steinman added that "[i]nfection with [cytomegalovirus] and/or [Epstein Barr Virus] are two possibilities for the GBS trigger"—presumably interpreting the evidence of Petitioner's URI as possibly establishing such an infection despite a lack of record evidence proving either's existence. But he ultimately proposed that the Tdap vaccine more likely explained Petitioner's GBS, "because there is a clear underlying mechanism to explain how the vaccine is the trigger" in comparison to those two wild infections. *Id.* at 7.

Next, Dr. Steinman outlined his theory for how the Tdap vaccine could trigger GBS. First, he noted that the Institute of Medicine (the "IOM") had evaluated the role that the alum adjuvant contained in the Tdap vaccine is believed to increase the vaccine's immunogenicity. First Steinman Rep. at 7–8; *Institute of Medicine, Adverse Effects of Vaccines: Evidence and Causality* (K. Stratton et al., eds., 2012), filed as Ex. 23 (ECF No. 48-1) ("2012 IOM Rep.").[7] In addition to describing the impact on immune response, it also noted that "alum may directly activate cells of the innate immune system through its effect on local inflammasome complexes leading to the release of inflammatory mediators." 2012 IOM Rep. at 88. Dr. Steinman's overall causation theory stemmed from the potentiality for this process to become aberrant.

[7] Dr. Steinman's First Report erroneously refers to this document as having been published in 2011. First Steinman Rep. at 7. More concerning is the fact that Petitioner has unhelpfully filed *the entire 800-plus* pages of the IOM 2012 Report, rather than only the specifically-relevant and cited selections. *See generally* Ex. 23. This is wasteful and unnecessary, and counsel in the future should show greater care in offering medical and scientific literature (which already in most Vaccine Program cases is filed excessively, and out of all proportion to its overall utility).

Dr. Steinman offered several additional items of literature confirming the immunogenic contributory role of alum as an adjuvant, although one item noted that "the basis for its adjuvanticity remains poorly understood." First Steinman Rep. at 8 (*quoting* H. Li et al., *Aluminum Hydroxide Adjuvants Activate Capsase-1 and Induce IL-1β and IL-18 Release,* 178 J. Immunol. 5271 (2007), filed as Ex. 33 (ECF No. 49-1)) ("Li"). He later noted that the 2012 IOM Report had questioned whether Tdap-like vaccines could be reasonably associated with GBS, but retorted that none of the literature he relied upon in this case had been considered in that report. First Steinman Rep. at 10–11; 2012 IOM Rep. at 587 (referencing ten studies, and concluding that "the mechanistic evidence regarding an association between diphtheria toxoid-, tetanus toxoid-, or acellular pertussis-containing vaccine and GBS" was inadequate). Dr. Steinman also cited the Tdap vaccine's package insert, which he noted admitted an increased risk of GBS after receipt of the vaccine—*if* the recipient previously experienced GBS within six weeks of a prior dose of a vaccine containing tetanus toxoid. First Steinman Rep. at 11; Adacel,[8] *Highlights of Prescribing Information*, https://www.fda.gov/downloads/biologicsbloodvaccines/vaccines/approvedproducts/ucm 142764.pdf (last visited Apr. 13, 2022) at 2, filed as Ex. 25 (ECF No. 48-3) ("Tdap package insert"). Notably, however, the Tdap package insert relies on an *older* IOM report not offered in this case (and presumably supplanted by the 2012 IOM Report filed by Petitioner, which itself only denies the ability to ascertain mechanistically if the vaccine is or is not causal). Tdap package insert at 2 n.1, 4.

Second, Dr. Steinman highlighted the importance of the fact (as Li had noted) that certain pro-inflammatory cytokines—in particular, IL-1β and IL-18—were activated in response to the alum adjuvant. First Steinman Rep. at 8; Li at 5275. IL-1β, he proposed, was "strongly unregulated during active GBS." First Steinman Rep. at 8; K. Nyati, et al., *Correlation of Matrix Metalloproteinases-2 and -9 with Proinflammatory Cytokines in Guillain-Barré Syndrome,* 88 J. Neurosci. R. 3540 (2010), filed as Ex. 34 (ECF No. 49-2) ("Nyati"). Another article also directly implicated IL-18 "in the pathogenesis of acute immune-mediated [peripheral nervous system] demyelination." S. Jander & G. Stoll, *Interleukin-18 is Induced in Acute Inflammatory Demyelinating Polyneuropathy,* 114 J. Neuroimm. 253 (2001), filed as Ex. 35 (ECF No. 49-3) ("Jander & Stoll"). And he noted that the predominant animal model used to study autoimmune peripheral demyelinating diseases like GBS, experimental autoimmune neuritis ("EAN"), had observed that treatments to suppress IL-18 seemed to in turn reduce autoantibody responses driving GBS—suggesting its importance to GBS's pathogenesis. A. Yu et al., *Neutralizing Antibodies to IL-18 Ameliorate Experimental Autoimmune Neuritis by Counter-Regulation of Autoreactive Th1 Responses to Peripheral Myelin Antigen,* 61 J. Neuropathol. & Experimental Neurol. 614 (2002), filed as Ex. 36 (ECF No. 49-4) ("Yu").

[8] Adacel is the trade name for the Tdap vaccine version administered in this case. Ex. 50.

Dr. Steinman deemed these articles to provide "a strong scientific foundation" for his theory. First Steinman Rep. at 9. But, on close review, they are less supportive than he assumed. Nyati, for example, noted that GBS's pathology in part requires breach of the blood nerve barrier by *both* immune cells specific to the disease's course (i.e. autoantibodies) as well as non-specific macrophages (immune cells that react to any pathogen without specificity). Nyati at 3540. A specific kind of enzyme is also involved in this breaching process, and Nyati's authors determined that the enzyme "can regulate the expression and activation of cytokines and hence play a complex role in inflammatory diseases" like GBS. *Id.* at 3541. Nyati looked specifically at the correlation between this enzyme level and levels of proinflammatory cytokines like IL-1β, finding the latter were higher during the later, progressive stages of the disease. *Id.* at 3545. But because the presence of these cytokines was observed in connection with a different immune process—and, more significantly, that this aspect of the process resulting in GBS was not deemed *instigative* of it (and indeed likely occurred after the disease process had already begun)—Nyati clearly does not stand for the proposition that merely increasing levels of IL-1β will *cause* GBS. At most, it is the *enzyme* studied in Nyati that promotes the cytokines at issue.

Jander & Stoll was similarly read by Dr. Steinman far more broadly than the actual article allows. Its authors deemed it to be a "widely accepted" concept that T-helper cells (which as a general matter encourage adaptive immune responses—for example, by aiding B cell production of antibodies specific to a particular pathogen) played an important role in GBS's pathogenesis, and that some T-helper cells were understood to cause upregulation of certain proinflammatory cytokines. Jander & Stoll at 253. At the same time, "[a]ntigen-presenting cells such as activated macrophages" were encouraging production of cytokines like IL-18, which in turn had the capacity to "favor a [T-helper cell]-like polarization of T cell-mediated immune responses." *Id.* Through both an animal study and small-sample measurement of the cytokine levels in 36 GBS patients, Jander & Stoll determined that there was "a role for macrophage-derived IL-18 in the pathogenesis of [T-helper cell]-mediated autoimmune demyelination in the [peripheral nervous system]." *Id.* at 257. This finding (like Nyati's) is thus far narrower than Dr. Steinman posits, as it observes increases in a relevant cytokine dependent on a factor *other* than initial vaccination, and moreover that influences a sub-step in the pathogenesis of GBS, as opposed to instigating the process at the outset. It says nothing about what *initially* causes the cytokine-promoting macrophages to react.

Yu is no different. Its authors relied on the EAN animal model to specifically consider "the function of IL-18 in T-cell-mediated EAN," noting at the outset that although high levels of IL-18 had been measured in GBS patients (relying for this proposition specifically from Jander & Stoll), but that not much was known about the role the cytokine plays in the course of autoimmune diseases. Yu at 614. Yu attempted to approach the question from an opposite end, evaluating what would occur if anti-IL-18 antibodies were used as therapy. *Id.* By finding that an anti-IL-18 treatment could effectively "attenuate" EAN, Yu's authors concluded they had illustrated the cytokine's importance to the disease, with some suggestion that the same ameliorative effect was

possible outside the context of the EAN experiment (which relies on use of an artificially-stimulated adjuvant to heighten the immune reaction, so that different variables can be tested). *Id.* at 615, 621. But, like Jander & Stoll, Yu's authors were focused on T-helper cells, based on the fact that "EAN is predominantly a T cell-mediated disease"—something not understood about GBS[9]—and hence Yu's findings were limited to how IL-18 upregulation interacts with T-helper cells and (in turn) "T cell-activation by antigen-presenting cells," or macrophages. *Id.* at 620.[10]

The remainder of Dr. Steinman's first report addressed the other two causation prongs that a Program petitioner must satisfy. He proposed that the second, "did cause" prong was met—but mainly in conclusory fashion, noting that literature insufficiently (in his view) provided a convincing mechanistic connection between certain wild virus infections (EBV or CMV) and GBS, and otherwise taking issue with the 2012 IOM Report's conclusion that the immune processes thought to drive GBS were not convincingly connected to the Tdap vaccine. First Steinman Rep. at 10–11. He also endorsed the onset of Mr. K.A.'s GBS as occurring in a medically-acceptable timeframe, when measured from date of vaccination. To do so, however, Dr. Steinman invoked an item of literature specific to the flu vaccine. First Steinman Rep. at 11; L. Schonberger et al., *Guillain-Barré Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976–1977,* 110 Am. J. Epidemiol. 105 (1979), filed as Ex. 42 (ECF No. 49-10) ("Schonberger"). Because Petitioner's onset occurred approximately three weeks[11] after vaccination, it was within Schonberger's observed interval. Schonberger at 109.

Second Report

By the time of the filing of his supplemental report, Dr. Steinman had the benefit of having reviewed the first report prepared by Respondent's Expert, Kathleen Collins, M.D. Thereupon began a "tit-for-tat," bickering exchange between the two that stretched over the next seven reports

[9] *Blackburn v. Sec'y of Health & Hum. Servs.,* No. 10-410V, 2015 WL 425935, at *15 (Fed. Cl. Spec. Mstr. Jan. 9, 2015) (peripheral neuropathies usually felt to be mediated by B cell-produced antibodies).

[10] Dr. Steinman also extensively discussed a study in which scientists suppressed IL-18 in EAN—but without a reduction in associated symptoms (thus undermining the contention that this cytokine was a central driver of pathogenesis). R. Duan et al., *IL-18 Deficiency Inhibits Both Th1 and Th2 Cytokine Production but Not the Clinical Symptoms in Experimental Autoimmune Neuritis,* 183 J. Neurolimm. 162 (2007), filed as Ex. 38 (ECF No. 49-6) ("Duan"). Duan's authors determined specifically a "net effect of no influence of IL-18 deficiency on EAN severity." Duan at 166. However, Dr. Steinman attempted to minimize Duan's significance, noting that it was a study performed on genetically-engineered, or "gene knockout" animals, and arguing that the findings in such a study were less useful or trustworthy, since he felt that medical science recognized that this technology had become "old and is now considered flawed." First Steinman Rep. at 10.

[11] Dr. Steinman somewhat erroneously identifies onset as occurring 18 days post-vaccination, or by August 30, 2013 (based on the August 12th vaccination)—even though the medical record states that around this time Petitioner sought treatment for URI symptoms only, with his neurologic complaints not beginning until five days later (September 4th). Ex. 9 at 6, 9, 11, 17. But since either date (August 30th or September 5th) is adequate from a temporal standpoint to fit Petitioner's timeframe theory (based on molecular mimicry as the causal mechanism), this difference does not meaningfully undermine Dr. Steinman's third prong opinion.

collectively filed by both experts. While for the most part their interaction sheds limited light on the contested issues, some reasonable elaborations or clarifications were provided—and as discussed below, eventually Petitioner (at the urging of the special master then responsible for the case) modified his theory entirely.

The first third of Dr. Steinman's supplemental report was spent quibbling with Dr. Collins's use of the term "influenza-like illness" in proposing a more likely cause for Petitioner's GBS. *See generally* Second Steinman Rep. at 1–3. Dr. Steinman deemed it insufficiently precise, and therefore not a reliable diagnosis upon which to base an argument about an alternative cause (despite the fact that the record in this case clearly establishes that Petitioner *was experiencing* a URI before his first manifestation of neurologic symptoms—even if no specific virus was identified).[12] He was not prepared to accept an illness that had not been specifically identified, by testing or otherwise, as an explanation for Petitioner's GBS, given the undeniable, identified "fact" of receipt of the Tdap vaccine. And he attempted an item-by-item rebuttal of literature Dr. Collins had referenced as contaminated by a similar amount of imprecision.

Turning to the central aspects of his theory, Dr. Steinman conceded that "[t]here is no epidemiologic proof that alum containing vaccines are related to GBS." Second Steinman Rep. at 3. But (block-quoting his own initial report), Dr. Steinman disputed the significance of this lack of proof, in light of the fact that no such epidemiologic evidence could *ever* completely "rule out" the possibility that a vaccine could be causal of GBS, or was in this case. *Id.* at 3–4. He noted that the 2012 IOM Report he had previously discussed in his first report had (when considering the epidemiologic evidence) stated only that the evidence was "inadequate to accept or reject" a Tdap-GBS association, leaving the matter open. *Id.* at 4. He also highlighted an item of epidemiologic evidence that in his view *allowed* for a potential Tdap association with acute disseminated encephalomyelitis ("ADEM"), a neuroinflammatory demyelinating disorder that impacts the brain and spinal cord (and hence somewhat comparable to GBS). R. Baxter et al., *Acute Demyelinating Events Following Vaccines: A Case-Centered Analysis,* 63 Clin. Infect. Diseases: An Official Publication of the Infectious Diseases Soc. of Am., 1456 (2016), filed as Ex. 44 (ECF No. 51-1) ("Baxter I"). Thus, Dr. Steinman maintained that *some* epidemiologic evidence did not rebut the possibility that an alum-adjuvanted vaccine could cause injury as he had proposed—even though Baxter I *does not address GBS*, a distinguishable peripheral neuropathy. Baxter I at 1456.

[12] Dr. Steinman also engaged in conduct I have in the past criticized him for: commenting on the standards governing Program cases, and elaborating on how he performs his role as expert, rather than simply providing the medical/scientific opinion for which he has been retained. *See,* e.g., *Rolshoven v. Sec'y of Health & Hum. Servs*., No. 14-439V, 2018 WL 1124737, at *21 (Fed. Cl. Spec. Mstr. Jan. 11, 2018). (Notably as well, in *Rolshoven* as here, Dr. Steinman's causation theory was based in part of the cytokine-stimulative impact of a vaccine's alum adjuvant. *Rolshoven,* 2018 WL 1124737, at *20). Indeed, he block-quoted in his second report an entire paragraph from a prior Program decision in support of his contention about the lack of weight to be given to the term "influenza-like illness." Second Steinman Rep. at 2. None of Dr. Steinman's reports in this case were prepared while I was presiding over this matter—but in future cases in which Dr. Steinman is retained that are assigned to me, *I will not compensate time spent on opinions on legal issues that he is not qualified to address.*

In other regards, Dr. Steinman merely vouched for the opinion he had previously offered. In reaction to Dr. Collins's contention that the individual items of literature he previously cited did not directly establish an alum/adjuvant-GBS association, Dr. Steinman protested that (a) the items offered were individually reliable, having been published in reputable journals, and (b) he reasonably relied on this evidence as links in his overall theory chain. Second Steinman Rep. at 4–6. Thus, articles like Li or Nyati were useful in *at least* demonstrating alum's impact on the immune process—and that it could trigger upregulation of cytokines significant to GBS's pathogenesis. *Id.* at 6–8.

Third Report

In reaction to the four-page rebuttal offered by Dr. Collins, Dr. Steinman offered 12 additional report pages—although discerning how the ongoing expert exchange was being expanded or improved upon with new, worthwhile points is exceedingly difficult.

First (and continuing his commentary on the legal standards relevant to the case's resolution—despite his admission that "I am not a lawyer" (Third Steinman Rep. at 1)), Dr. Steinman spent a considerable amount of time maintaining that the term "influenza-like illness" could not credibly be employed, given its vague character, to establish a persuasive alternative explanation for Petitioner's GBS. Third Steinman Rep. at 1–2. He deemed the known fact of Petitioner's receipt of the Tdap vaccine to outweigh the unestablished possibility of an intercurrent infection that could not be precisely identified (even though the fact of Petitioner's URI symptoms cannot be contested). *Id.* at 2.

Second, Dr. Steinman objected to Dr. Collins's general contention that his overall theory was nothing more than a series of loosely-connected, "cherry-picked" studies that were inconsistent overall (in part because they were a combination of animal and human studies with differing and distinguishable methodologies). Third Steinman Rep. at 2–3. After expressing umbrage at the use of the term "cherry picking" generally (and emphasizing his vast expertise testifying in Vaccine Program cases—and the attendant knowledge he presumably had gained from how claims should be evaluated, in light of the fact that vaccine injury causation could never be determined to any degree of scientific certainty), Dr. Steinman endeavored to explain how the individual components of his theory connected. *Id.* at 3–4.

He thus reiterated that alum had been demonstrated by reliable science to upregulate certain pro-inflammatory cytokines (albeit in the context of promoting immunogenicity)—but also that it could do so even *outside* the context of experiments relying on the use of additional stimulative agents to enhance the response (the reason Dr. Collins had criticized the invocation of such other evidence). Third Steinman Rep.at 5; J. Mannhalter et al., *Modulation of the Human Immune*

Response by the Non-Toxic and Non-Pyrogenic Adjuvant Aluminum Hydroxide: Effect on Antigen Uptake and Antigen Presentation, 61 Clin. Exp. Immunol. 143, 144 (1985), filed as Ex. 47 (ECF No. 54-2) ("Mannhalter") (evaluating the influence of the adjuvant on the immune process by *in vitro* testing of vaccinated blood samples, adding tetanus toxoid antigens to the samples).

Dr. Steinman further underscored his prior contention that a number of human studies "covered the effect of alum on the human immune response," based on blood testing from individuals diagnosed with GBS. Third Steinman Rep. at 6. He included a block quote from Li, followed by several lengthy cites to Dr. Collins's second report, in an effort to bolster the reliability of this part of his opinion. *Id.* at 6–7. He did the same with Jander & Stoll, and added even more lengthy block quotes from his prior reports as support (rather than offering new commentary based on additional scientific support not previously referenced or highlighted). *Id.* at 7–8. And he repeated the view that articles like Yu (demonstrating suppression of IL-18 via antibody treatments in animal studies) were better evidence of the association of the cytokine to the demyelination characteristic of GBS than animal experiments involving gene "knockout" (which he had previously acknowledged produced results contrary to his primary contentions). *Id.* at 8–9.

Fourth Report

Dr. Steinman's fourth report arrived three years into the claim's life—and also after the special master previously presiding over the case had expressed the willingness to entertain a revised causation theory (since Respondent was deeming the adjuvant-based theory to be implausible).[13] This report thus pivoted to a "new" theory, albeit one that Dr. Steinman has offered repeatedly in Vaccine Program cases past and present: that the autoimmune process leading to GBS was instigated by the Tdap vaccine via the mechanism of molecular mimicry.

Dr. Steinman began his foray into this second/alternative causation theory by briefly vouching for his own expertise, in both the fields of immunology as well as neuroinflammatory autoimmune disease. Fourth Steinman Rep. at 1. He represented that his prior theory was not "mutually exclusive" from his new theory, which focused on "how the contents of the [Tdap] vaccine a) trigger via molecular mimicry immune responses to myelin components that are known to be targeted in GBS." *Id.*

First, Dr. Steinman listed the specific antigenic components of the Tdap vaccine. Fourth Steinman Rep. at 2–3. Second, he turned to a brief review of the general concept of molecular mimicry, in which "shared structures on a virus or bacteria or in a vaccine can trigger a cross-reactive response to self." *Id.* at 3 (citations omitted). Such sharing, or "homology," is established by showing amino acid (the building blocks of protein) identity (whether or not sequential)

[13] *See* Section III below, discussing the case's procedural history.

between the foreign presenting antigen and the self structure. If antigen and self structure are sufficiently similar, then antibodies produced by the immune system in reaction to the foreign antigen (and intended to attack it) will *also,* if mistakenly, attack self. But because homology is common in nature (given the total limited number of amino acids that constitute proteins), it is important to focus on homology specific to the "disease-related" situs for the cross-reactive attack. *Id.* at 4.[14] In the case of GBS, it is understood that the relevant situs are ganglioside structures found on the surface of myelin basic protein molecules. *Id.*

Dr. Steinman thereafter engaged in the same overall mimicry showing that characterizes most of his expert reports in which the theory is offered. *See generally* Fourth Steinman Rep. at 5–16. Specifically, he (a) attempted to propose the degree of amino acid homology that would be necessary, arguing that five (and perhaps even four) out of a twelve amino acid string is sufficient (*Id.* at 5–8) (b) performed "BLAST" searches[15] with an online government database for the antigenic components of the Tdap vaccine, in order to identify the amino acids comprising them (*Id.* at 8–10), and (c) compared them to identified GBS targets. *Id.* at 10–15. Dr. Steinman also compared the amino acids found in the protein components of the vaccine's tetanus toxoid and diphtheria toxin components with those making up neurofascin, a nerve protein expressed at certain nerve nodes believed to be situses for demyelinating autoimmune attack (although more specifically associated with a distinguishable peripheral neuropathy, chronic inflammatory demyelinating polyneuropathy ("CIDP")). *Id.* at 9–13; J. Devaux et al., *Neurofascin-155 IgG4 in Chronic Inflammatory Demyelinating Polyneuropathy*, 86 Neurology® 800, 800 (2016), filed as Ex. 63 (ECF No. 63-3). He concluded from the foregoing that "a compelling case" existed in support of his contention that "molecular mimics in the [Tdap] vaccine . . . triggered GBS due to homologies with antigens that are homologous mimics of the vaccine and which are targeted in this neuroinflammatory condition." Fourth Steinman Rep. at 17.

Dr. Steinman's fourth report provided little else to substantiate this alternative/new causation theory, however, outside his detailed review of the molecular biology data supporting his contentions. He offered only a single additional paragraph, maintaining that "immunity to nervous system antigens like myelin is rather widespread in normal individuals"—presumably conceding (comparable to his earlier admission about the common nature of homology) that a pathogenic cross-reaction between antigens and self, driven by antibodies, is uncommon. Fourth Steinman Rep. at 17. As a result, "[o]ther genetic and environmental factors are necessary before these self-reactive immune responses to myelin might trigger GBS"—but Dr. Steinman provided

[14] Dr. Steinman's Fourth Report includes verbatim citations to charts and block-quotes from articles that appear in nearly all reports he offers in the Program when molecular mimicry is alleged as a causal theory—and therefore I do not cite them herein.

[15] Basic Local Alignment Search Tool ("BLAST") is a medical/scientific internet resource that assists researchers in finding regions of similarity between biological sequences of amino acids. The program compares nucleotide or protein sequences to sequence databases and calculates the statistical significance. BLAST, U.S. National Library of Medicine, https://blast.ncbi.nlm.nih.gov/Blast.cgi (last visited Apr. 13, 2022).

nothing to identify what they might be, either theoretically or in Petitioner's specific case. *Id.* And he referenced no other literature or independent support for his contention that the Tdap vaccine could cause GBS via molecular mimicry.

Fifth Report

Dr. Steinman's last report concluded the expert exchange—aalthough it reflects the same bickering quality, or wholesale reproduction of prior arguments, that characterizes most of his prior reports filed in this case. Thus, the first whole page of the final report represented Dr. Steinman's renewed reaction to Dr. Collins's "cherry picking" contention. Fifth Steinman Rep. at 1–2. He also reproduced sections or charts from his prior reports, such as the schematic contained in his fourth report showing how Dr. Steinman's BLAST search for amino acid homologies with self structures supports causation. *Id.* at 11.

Nevertheless, Dr. Steinman did make some salient points bearing on his opinion (and regarding both proposed causation theories). First, he reacted to Dr. Collins's reference to one article as unsupportive of the alum adjuvant causation theory. Fifth Steinman Rep. at 2; A. Linneberg et al., *Association of Subcutaneous Allergen-Specific Immunotherapy with Incidence of Autoimmune Diesease, Ischemic Heart Disease, and Mortality,* 129 J. Allergy Clin. Immunol. 413 (2012), filed as Ex. D, Tab 3 (ECF No. 57-3) ("Linneberg"). Dr. Steinman noted that Linneberg's focus was on allergic disease, which meant it evaluated individuals with "immune responses biased towards what is called Th2" cells—a kind of specialized T-helper cell.[16] Fifth Steinman Rep. at 2. But because "[m]ost autoimmune diseases are considered [mediated by] Th1" cells,[17] Linneberg's alum findings reflected "a poor starting point for assessment of whether alum could increase propensity to GBS or to any autoimmune disease." *Id.* at 2–3.

Dr. Steinman went on to represent (again) that Dr. Collins's embrace of an "influenza-like illness" as causal of Petitioner's GBS was medically and scientifically imprecise. Fifth Steinman Rep. at 3–6. There was no evidence of what precisely had caused Petitioner's URI symptoms—and the specific kinds of infections associated with GBS (CMV, EBV, etc.) were not demonstrated present either, *Id.* at 3–4. The proposed alternative cause was to Dr. Steinman inadequately

[16] The T-helper cell encourages the production of antibodies to antigens that interact with B-cells. *Helper Cells*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=64157 (last visited Apr. 13, 2022).

[17] Notably, this contention is somewhat in tension with (a) the greater likelihood that peripheral neuropathies like GBS are B-cell mediated (i.e. through production of cross-reactive autoantibodies), and (b) the fact that the molecular mimicry theory espoused in this case depends on vaccine antigens (which are intended to "teach" the immune system to recognize the antigen when presented in wild form—through the production of antibodies—by stimulating B cells) cause the production of cross-reactive autoantibodies. *This is the intended mechanism of the tetanus component of the vaccine. See* Tdap package insert at 3 ("[]rotection against disease is due to the development of neutralizing antibodies to tetanus toxin"). It thus reflects Dr. Steinman's tendency to shift his argument, back and forth from B to T cells, depending on how he is questioned or what he wishes to emphasize. *See,* e.g., *Blackburn,* 2015 WL 425935, at *29.

established. Otherwise, even the formal definition of "influenza-like illness" was no more than an "unconfirmed" diagnostic description, lacking in the precision needed to embrace it as more likely causal than the known fact of vaccination. *Id.* at 5–6.

Other aspects of Dr. Collins's argument, as highlighted in her final report, also received Dr. Steinman's criticism. One item of epidemiology, for example (sharing many of the same authors as Baxter I) was deemed flawed by Dr. Steinman, since its own authors admitted that the possibility of a vaccine-GBS association could not be excluded. Fifth Steinman Rep. at 7; R. Baxter et al., *Lack of Association of Guillain-Barré Syndrome with Vaccinations,* 57 Clin. Infect. Diseases: an Official Publication of the Infectious Diseases Soc. of Am. 197, 203 (2013), filed as Ex. A, Tab 6 (ECF No. 37-6) ("Baxter II"). The 2012 IOM Report similarly kept the door open to an association between Tdap and GBS, deeming the evidence for or against a causal relationship ultimately equivocal. Fifth Steinman Rep. at 8–9. And in any event, the IOM's analytic purposes and standards for evaluating vaccine safety generally were far higher than "the one relevant to determining Vaccine Act causation." *Id.* at 10.

B. Kathleen Collins, M.D., Ph.D.

Dr. Collins submitted four expert reports on behalf of Petitioner. Report, dated Nov. 28, 2017, filed as Ex. A (ECF No. 36-1) ("First Collins Rep."); Report, dated June 8, 2018, filed as Ex. C (ECF No. 43-1) ("Second Collins Rep."); Report, dated May 6, 2019, filed as Ex. D (ECF No. 56-1) ("Third Collins Rep."); Report, dated Jan. 23, 2020, filed as Ex. E (ECF No. 66-1) ("Fourth Collins Rep.").

As shown in her CV, Dr. Collins received her B.A. from Wellesley College and her M.D. and Ph.D. from John Hopkins, University School of Medicine. Ex. B (ECF No. 36-2) (Dr. Collin's Curriculum Vitae ("Collins CV")) at 1. She then had postdoctoral training in Internal Medicine at the Brigham and Women's Hospital, Infectious Disease Clinical Fellow, Research Fellow at Harvard University, and a Postdoctoral Fellowship at the Massachusetts Institute of Technology. *Id.* Dr. Collins was a professor of Microbiology and Immunology at the University of Michigan's Medical School. *Id.* She has conducted several studies on HIV, with publication of additional studies pending. *Id.* at 5–7. She also currently has graduate students and postdoctoral fellows she is training. *Id.* at 10. Dr. Collins has given several presentations on the topic and similar topics. *Id.* at 14–17. She has published several articles and reviews/book chapters on the topics of HIV, treatment of HIV, infection, and reactivation. *Id.* at 18–23.

First Report

Dr. Collins first summarized the materials she reviewed in preparing her report and recounted the medical facts specific to Mr. K.A.'s case. First Collins Rep. at 1–4. In so doing, she

emphasized that Petitioner's treaters "consistently attributed the GBS to a prior respiratory infection," citing examples from the record. *Id.* at 2. She also briefly defined GBS, describing its pathogenesis of autoimmunity consistent with what Dr. Steinman proposed, and noting also the association between GBS and the *Campylobacter jejuni* bacterial infection. *Id.* at 4.

Next, Dr. Collins reviewed Dr. Steinman's causation theory, noting that he relied on the alum adjuvant as causal—but that the scientific and medical evidence he cited in support did not actually "demonstrate that alum causes GBS," and otherwise was not even relevant to his contention. First Collins Rep. at 4. Li, for example, actually demonstrated that "[a]lum, when used by itself, did not stimulate cytokine production" by the immune cells that were studied, and thus was not supportive of the alum's role proposed by Dr. Steinman. *Id.* at 4–5; Li at 5276 ("[o]ur results clearly show that Alum by itself is incapable of promoting IL-1β transcription," and "aluminum particles were reported to be unable to activate the inflammasome" alone). Rather, the stimulative effect of alum was dependent upon the introduction of other experimental agents, diminishing the conclusion that the alum alone could be pathogenic in the manner proposed. First Collins Rep. at 5.

The same distinctions could be drawn for other items of literature that Dr. Steinman cited. Nyati, Dr. Collins observed, only concluded that certain levels of proinflammatory cytokines (including IL-1β) were higher at different phases of GBS's progression—not that alum caused their specific upregulation or otherwise drove the disease process at the outset. First Collins Rep. at 5; Nyati at 3543–45. Jander & Stoll similarly only observed increased levels of IL-1β in studied animals and a small human sample of GBS patients, but made no determinations about the propensity of vaccine adjuvants to trigger the condition. First Collins Rep. at 5; Jander & Stoll at 257. And three other animal studies that relied on inducing demyelinating disease only provided further evidence that the cytokines identified by Dr. Steinman played a role in the progression of disease (although the studies relying on genetically-altered animal subjects—which Dr. Steinman went to great lengths to distinguish (*see* footnote 10, above)—seemed to undermine the conclusion about the significance of IL-18). First Collins Rep. at 5–6. Overall, Dr. Collins interpreted these articles and studies to provide no support for his contention that alum in the Tdap vaccine can produce GBS, even if the identified cytokines *themselves* can be harmful at certain stages in the disease's progression if elevated. *Id.* at 6. Rather, there was more reliable support for viral or bacterial infections as a trigger. *Id.* And in so asserting, Dr. Collins referenced items of literature filed by Dr. Steinman that supported this conclusion. *Id* (citations omitted).[18]

[18] Dr. Collins even took issue with Dr. Steinman's Schonberger reference in support of a flu vaccine/GBS link, arguing that the association "was specific to the 1976 vaccination," and hence inherently limited—and in any event that the version of the vaccine then in use was not shown to include alum as an adjuvant. First Collins Rep. at 6. In fact, it is the case that the version of the flu vaccine administered in the U.S. generally does not contain an adjuvant. *See* Centers for Disease Control and Prevention, *Vaccine Adjuvants,* https://www.cdc.gov/vaccinesafety/concerns/adjuvants.html (last visited Apr. 14, 2022), filed as Ex. A, Tab 13 (ECF No. 38-3) ("Vaccine Adjuvants"), at 1–2 (explaining the vaccines that usually include adjuvants). Only a recently-licensed version of the flu vaccine contains an adjuvant. Vaccine Adjuvants at 2.

Dr. Collins also pointed to other literature evidence suggesting an adjuvant like alum would not likely be causal of disease. She noted that the majority of vaccines include some kind of adjuvant, with aluminum particularly common. First Collins Rep. at 6; Centers for Disease Control and Prevention, *Vaccine Adjuvants,* https://www.cdc.gov/vaccinesafety/concerns/adjuvants.html (last visited Apr. 14, 2022), filed as Ex. A, Tab 13 (ECF No. 38-3), at 1–2 (aluminum adjuvant found in Hepatitis A and B, DTaP/Tdap, Hib, HPV, and pneumococcal vaccines). But reliable studies had found no association between many of these adjuvanted vaccines and autoimmune disorders, including GBS. First Collins Rep. at 7; G. Deceuninck et al., *Absence of Association Between Guillain-Barré Syndrome Hospitalizations and HPV-Vaccine*, Expert Review of Vaccines, (2017), filed as Ex. A, Tab 4 (ECF No. 37-4) (no increase in incidence of GBS after receipt of HPV vaccine, based on 100 cases analyzed).

A larger study that looked more specifically at the possibility of a Tdap-GBS relationship (among a large number of vaccines) found the same absence of an association. *See generally* Baxter II. Baxter II began with a sample pool of the more than three million members of Kaiser Permanente of Northern California, an integrated healthcare delivery system. Baxter II at 198. From that pool, Baxter II's authors identified 415 individuals between 1994 and 2006 who experienced GBS, based on accepted diagnostic definitions of the disease. *Id.* at 199–200. Of that total, only 25 patients had even *received* a vaccine within six weeks of symptoms onset—and only three of that subset had received a tetanus-containing vaccine. *Id.* at 200. It also employed a "case-centered" analysis,[19] observing no statistically-significant increased incidence of post-Tdap GBS. *Id.* at 200–02.[20]

Finally, Dr. Collins proposed that the medical record better supported the conclusion that Petitioner's GBS was attributable to the "influenza-like illness" that he appeared to have experienced in the interval between his vaccination and onset of neurologic symptoms. First Collins Rep. at 7. CMV and EBV infections were in particular associated with GBS, although, as Dr. Collins admitted, the record was a bit more equivocal on identifying either as present in Petitioner's case. *Id.* Regardless, there was literature support connecting URI-type infections with GBS. *See,* e.g., C. Tam et al., *Guillain-Barré Syndrome and Preceding Infection with Campylobacter, Influenza and Epstein-Barr Virus in the General Practice Research Database,*

[19] Baxter II defines this to be an analysis in which researchers "look back from the date of onset of the [relevant] adverse event and determine whether there is a clustering of vaccinations (exposures) in the risk interval prior to onset." Baxter II at 198. The methodology is "best suited to outcomes hypothesized to have an acute onset during a transient period of increased risk after exposure"—a "risk interval." *Id.* The intervals used in Baxter II—six weeks and ten weeks—are consistent with Petitioner's onset and Dr. Steinman's timeframe contentions.

[20] In conducting their case-centered analysis, Baxter II's authors noted that within the study's three million-plus patient population in the relevant Kaiser Permanente Northern California care group, approximately 1.9 million doses of DTaP (the childhood version of Tdap) were administered, with *no* observed instances of GBS in any risk interval (as compared to one instance of post-Tdap GBS—underscoring the significance of a lack of association. Baxter II at 200.

PLoS One e344 (2007), filed as Ex. E, Tab 3 (ECF No. 66-4) ("Tam"); J. Stowe et al., *Investigation of the Temporal Association of Guillain-Barré Syndrome with Influenza Vaccine and Influenzalike illness using the United Kingdom General Practice Research Database,* 169 Am. J. Epidemiol. 382 (2008), filed as Ex. E, Tab. 23 (ECF No. 66-24) ("Stowe"); H. Lehmann et al., *Guillain-Barré Syndrome After Exposure to Influenza Virus*, 10 Lancet Infect. Dis. 643, 644–45 (2010), filed as Ex. A, Tab 10 (ECF No. 37-10) (review article taking into account Tam, Stowe, and other items of literature, and observing increased incidence of GBS after influenza-like illnesses).

Second Report

Dr. Collins prepared a second report to respond briefly to Dr. Steinman's rebuttal points in his own second report. Regarding his contention that "influenza-like illness" was too vague of a basis for a finding of an alternative cause for Petitioner's GBS, she proposed that the term was in her experience commonly employed to describe "the symptoms caused by a [URI]," adding that it was often difficult to identify through testing a precise viral cause for such symptoms. Second Collins Rep. at 1. In addition, Dr. Collins deemed it significant that reliable literature she had offered to support the association between GBS and a variety of wild virus infections had also employed the term, suggesting it had more general support than Dr. Steinman argued (and noting other instances where reputable studies utilized it). *Id.* at 1–2 (citations omitted).

Dr. Collins next reiterated her prior point that a number of reliable epidemiologic studies showed no association between alum-adjuvanted vaccines (including Tdap) and GBS. Second Collins Rep. at 2–3. The fact that Baxter I had observed a potential relationship between *ADEM* and Tdap was overshadowed by Baxter II's explicit findings of a *lack* of an association with the relevant injury, GBS. *Id;* Baxter II at 203. In response, Dr. Steinman had shown no reasoned grounds for disputing these findings, and even conceded he could offer no epidemiologic proof in response. Second Collins Rep. at 3. Instead, he had (in Dr. Collins's words) "'cherry picked' minor results from different papers utilizing disparate experimental systems to form his hypothesis"—resulting in a theory that was, even taken in total, weak. *Id*. Thus, he over-relied on *in vitro* studies associating alum and cytokine increases that the 2012 IOM Report had deemed not wholly worthwhile for present purposes, while emphasizing the role of cytokine upregulation in promotion of GBS generally—even though the IOM had expressly questioned "the oversecretion of cytokines as an operative mechanism" for vaccine-instigated disease. 2012 IOM Report at 76.

Third Report

Dr. Collins began her third report by highlighting some of the medical record evidence establishing that in fact Mr. K.A. had experienced the kinds of symptoms that would be associated with an "influenza-like illness," as the term is defined by the Centers for Disease Control, adding that the explanatory power of this evidence in identifying an etiology for Petitioner's GBS "did

not require identification of a specific organism," but could instead be supported merely by the "complex of symptoms that Mr. K.A. experienced." Third Collins Rep. at 1. She emphasized that the association between a wild influenza infection and GBS was better-established than between the flu vaccine and GBS. *Id.;* F. DeStefano et al., *Principal Controversies in Vaccine Safety in the United States,* Clinical Infectious Diseases: An Official Publication of the Infections Diseases Society of America (Stanley Plotkin, ed. 2019), filed as Ex. D, Tab 1 (ECF No. 57-1) ("DeStefano"). Thus, Petitioner's established URI better explained the cause of his GBS than the Tdap vaccine, despite the fact that the specific nature of his infection could not be ascertained.

Next, Dr. Collins again reviewed her contentions that direct evidence she had offered *refuted* Dr. Steinman's theory that alum-adjuvanted vaccines could cause GBS—since the studies she offered demonstrated no increase of GBS post-vaccination. Third Collins Rep. at 2. Indeed, DeStefano referenced additional studies that undercut directly a connection between autoimmunity and aluminum adjuvants. DeStefano at 4–5; Linneberg. Linneberg's authors considered a sample of 18,000 patients who received specific kind of injection-based treatment (containing aluminum hydroxide as an adjuvant) for allergies, but experienced a lower incidence of autoimmune-mediated diseases than the controls, suggesting at least that the alum adjuvant was not generally pathogenic. Linneberg at 416, 418. Of course (and as Dr. Steinman observed), Linneberg's focus is distinguishable from the issues raised by this case.

In Dr. Collins's view, because Dr. Steinman could not identify support for his theory from literature directly testing his hypothesis (and indeed, in her view such literature undermined his theory), he instead engaged in the selective invocation of scientific evidence she had previously criticized. Third Collins Rep. at 2. Mannhalter, for example, did observe a heightened cytokine (as well as cellular immune, in the form of T cells) response in human sera that had received an aluminum-adjuvanted tetanus toxoid-containing vaccine (in comparison to subjects who only received tetanus toxoid alone)—but did not say anything about the capacity of the vaccine to *cause* GBS as a result of this increase. Collins Third Rep. at 2–3; Mannhalter at 149. In fact, Mannhalter's authors explicitly observed that the aluminum adjuvant was "free of side effects" like toxicity or antigenicity, when compared to bacterial adjuvants, underscoring that the adjuvant was unlikely to be disease-causing in the manner proposed by Dr. Steinman. Collins Third Rep. at 3; Mannhalter at 150 (noting the benefits of an aluminum hydroxide adjuvant over a bacterial-based adjuvant).

Dr. Collins then summarized (for the second time) the literature offered by Dr. Steinman, pointing out that no specific article individually established that the adjuvant components of the Tdap vaccine could cause GBS, or even the lesser contention that certain cytokine increases "are associated with the use of aluminum containing vaccines in humans." Collins Third Rep. at 4. In reaction to Dr. Steinman's protestations that a more "perfect experiment" directly establishing the proposed causal link was impossible (and thus that he was forced to rely on a chain of indirect determinations), Dr. Collins noted that actually a number of studies *had looked directly* at the

results of receipt of adjuvant-containing vaccines, but observed no increase in autoimmune disorders (although some studies were not specific to the Tdap vaccine). *Id;* T. Verstraeten et al., *Analysis of Adverse Events of Potential Autoimmune Aetiology in a Large Integrated Safety Database of AS04 Adjuvanted Vaccines,* 26 Vaccine 6640 (2008), filed as Ex. A, Tab. 3 (ECF No. 37-3) (observing no association between HPV and HBV vaccines with aluminum-component adjuvant and large number of autoimmune conditions, including GBS; more than 60,000 subjects considered).

Regarding Dr. Steinman's arguments about the Tdap vaccine package insert, Dr. Collins observed that the insert's seeming embrace of a tetanus toxoid-oriented GBS risk was based upon a 1994 IOM Report, which she deemed superseded by subsequent determinations. Third Collins Rep. at 5–6; Tdap Package Insert at 2, 4 n.1. Moreover, Baxter II (published in 2013) was in her view (as previously argued) reliable epidemiologic proof establishing no evidence of an increased risk of GBS after the Tdap vaccine. Third Collins Rep. at 6. DeStefano had embraced this finding. Third Collins Rep. at 6–7; DeStefano at 4 (referencing Baxter II). Thus, the bases for the package insert's warnings were well outdated (and in any event GBS was not even included as a potential adverse event for an individual who had *not* been previously diagnosed with it post-vaccination). Third Collins Rep. at 7; Tdap package insert at 2, 4 n.1 (relying on 1994 IOM Report).

Fourth Report

Dr. Collins's final report reacted to the second causation theory embraced by Dr. Steinman, involving molecular mimicry between Tdap antigenic components and nerve structures associated with GBS. She acknowledged that Dr. Steinman was able to establish via animal models the *potentiality* of molecular mimicry as driving certain autoimmune diseases, like ADEM, but denied that this was helpful in establishing that the same kind of process was likely to cause GBS in a human. Fourth Collins Rep. at 9. In addition, while Dr. Collins did not dispute that Dr. Steinman had demonstrated homology between certain of the Tdap vaccine's antigenic components and self nerve-associated structures, she contended that this was insufficient by itself to establish molecular mimicry as the likely pathogenic mechanism, given how often homology occurred in nature without causing disease. *Id;* 2012 IOM Rep. at 70.

Dr. Collins otherwise revisited some of her earlier points, although she emphasized aspects of her argument that were less highlighted in previous reports. For example, she noted again some of the wild infections, viral and bacterial, that are known to be associated with GBS—*Campylobacter jejuni*, CMV, and EBV. Fourth Collins Rep. at 2–5. Although all were understood to be likely GBS triggers, they could not be said in each case to be mediated via molecular mimicry (thus diminishing the overall value of that as an explanatory mechanism herein for GBS across-the-board).

A *Campylobacter* bacterial infection, for example, was deemed likely to cause a particular kind of GBS—acute motor axonal neuropathology ("AMAN")—via autoantibodies produced as a result of molecular mimicry. Fourth Collins Rep. at 2. But this GBS variant was distinguishable from the most common form experienced in the U.S., AIDP (likely the version Mr. K.A. suffered from), the triggering pathogenesis of which was less well understood. *Id.* at 2–3. CMV infections were also associated with GBS, but studies regarding antibodies once believed to drive GBS after a CMV infection had "not borne out a role for molecular mimicry" under such circumstances, whereas other immune cells (notably T cells) not likely attributable to molecular mimicry *did* seem pathogenic in such circumstances. *Id.* at 3; D. Orlikowski et al., *Guillain-Barré Syndrome following Primary Cytomegalovirus Infection: A Prospective Cohort Study*, 52 Clin. Infect. Diseases 837, 843 (2011) filed as Ex. E, Tab 12 (ECF No. 66-13) ("Orlikowski") (impact of CMV infection itself, and associated viral replication, more likely to drive CMV-caused GBS than antibody response to infection; antibody response was more associated with secondary reaction to infection).

Dr. Collins also emphasized again her opinion that an influenza-like illness was a far more likely cause of Petitioner's GBS in this case. Despite the lack of definitive proof identifying Petitioner's precise infection, Petitioner's memorialized symptoms were evidence of a URI that reasonably could be considered an influenza-like illness. Fourth Collins Rep. at 4–5. She noted as well that literature she had filed specifically included the category "influenza-like illness" as a pre-GBS causal risk factor—contrary to Dr. Steinman's assertion that the term/concept was unscientifically vague. *Id.* at 5–7; Tam at 5 (noting a previously-unreported "18-fold increased risk of GBS in the two months following [an influenza-like illness]," based on a case-control study involving ten years of data for UK GBS patients). Other literature stood for the same contention. Fourth Collins Rep. at 6–8; Stowe at 4 (out of 690 studied individuals, 99 reported a pre-GBS onset influenza-like illness; no greater incidence of GBS post-flu vaccination seen in comparison to greater risk after influenza-like illness); L. Grimaldi-Bensouda et al., *Guillain-Barré Syndrome, Influenzalike Illnesses, and Influenza Vaccination During Seasons with and without Circulating A/H1N1 Viruses*, 174 Am. J. Epidemiol. 326 (2011), filed as Ex. E, Tab 25 (ECF No. 66-25) (greater risk of GBS post-influenza-like illness).[21]

III. Procedural History and Parties' Arguments

[21] In fact, a different study showed that receipt of a specific version of the flu vaccine was associated with a *reduced* risk of GBS. Fourth Collins Rep. at 8; C. Vellozzi et al., *Cumulative Risk of Guillain-Barré Syndrome Among Vaccinated and Unvaccinated Populations During the 2009 H1N1 Influenza Pandemic*, 104 Am. J. Pub. Health 696, 698 (2014), Filed as Ex. E, Tab 25 (ECF No. 66-26).

A. Procedural History

As noted, the case was filed in August 2016, and initially assigned to a different special master. After the filing of records was completed, Respondent filed his Rule 4(c) Report opposing compensation in February 2017. ECF No. 21. Over the next two years, the parties began filing expert reports, and the back-and-forth between Drs. Steinman and Collins over Petitioner's initial causation theory described above began.

Notably (and after the case had existed for nearly three years), it was discussed during a July 2019 status conference (perhaps in reaction to questions about whether the case was likely to settle) that Respondent deemed Petitioner's causation theory implausible—prompting the special master formerly presiding over the case to order a deadline for Petitioner to file a new report, but this time "modifying" the theory to allege molecular mimicry as the causal mechanism. Scheduling Order, dated July 19, 2019 (ECF No. 59). Dr. Steinman did so by that fall, and another, shorter round of expert report filings occurred, with the final report (from Dr. Steinman) filed in June 2020.

The case was subsequently reassigned to me in January 2021. ECF No. 79. I proposed that the case be resolved on the record, and set a schedule for briefs. The final brief was filed in October 2021, and the matter is now ripe for decision.

B. Briefs For and Against Causation

Motion for Entitlement

Petitioner's brief in favor of entitlement included several lengthy excerpts from the record and from Dr. Steinman's prior reports, but it also summarized his arguments for why he should be found to have preponderantly established his burden of proof. First, Petitioner maintained under either "sound and reliable" theory proposed by Dr. Steinman that the "can cause" prong had been established (although Petitioner seemed to stress the newer molecular mimicry theory over the theory relying on the adjuvant). Mot. at 23–31, 45–47. Dr. Steinman, he maintained, was eminently qualified to offer the molecular mimicry, and the methodology he employed to explain how antigenic components of the Tdap vaccine could cause a cross-reaction in the nerves leading to GBS was based on reliable and accepted science. *Id.* at 45–47.

The other two causation prongs were also satisfied, Petitioner argued. The second, "did cause" prong was established because no alternative viral cause had been identified, and some treaters had speculated that his GBS could have a vaccine association. Mot. at 48. The timeframe for his symptoms onset, beginning in 18–19 days post-vaccination, was consistent with accepted

literature like Schonberger for how long it would take for an autoimmune process driven by molecular mimicry to begin and to start manifesting symptoms. *Id.* at 49–50. Nor, Petitioner argued, did Respondent disagree that the timeframe was acceptable. *Id.* at 50. And Respondent had not otherwise carried his burden (assuming a burden shift) to establish a "factor unrelated"—here, the unspecified "influenza-like illness"—as more likely causal of Mr. K.A.'s GBS. *Id.* at 50–52.

Opposition

Respondent's opposition to entitlement contests all of the above. Regarding the first causation prong, he maintains that Dr. Steinman's adjuvant-based theory is "speculative and premised on untenable leaps in logic that lack reliable medical support." Opp. at 19. Reiterating some of Dr. Collins's criticisms, Respondent notes that the items of literature offered either were isolated experimental incidents (and hence too artificial to say anything meaningful about how alum's adjuvant role would cause a pathogenic cytokine response *in vivo*), or inadequately linked to GBS's instigation. *Id.* at 20–21. Dr. Steinman also acknowledged he lacked persuasive direct proof, relying instead on the vaccine's package insert—despite the low probative value given to that kind of evidence in the Program. *Id.* at 22. And the timeframe for such a cytokine-driven pathologic process would be far shorter than the 18 days in this case. *Id.* at 23, *citing Montgomery v Sec'y of Health & Hum. Servs.,* No. 15-1037V, 2019 WL 2511352, at *5 (Fed. Cl. Spec. Mstr. May 21, 2019) (theory relying on alum to spark cytokine reaction would require evidence of close-in-time reaction to vaccination—not onset occurring 10 or more days post-vaccination).

The second theory, relying on molecular mimicry as the proposed mechanism, was in Respondent's estimation no more successful. Although Respondent conceded that "the general concept of molecular mimicry is an accepted theory of causation for human autoimmune disease under certain circumstances," he argued that it had not *in this case* been shown to explain persuasively how the Tdap vaccine could lead to GBS. Opp. at 25. In particular, this was because Dr. Steinman had minimized the likely role Petitioner's intercurrent URI had played in causing GBS. *Id.* There was ample record evidence of the infection and the symptoms it spawned before GBS onset, and the mere fact that the infection had not been precisely identified did not matter. *Id.* at 26–28.[22] Otherwise, the mere showing of homology alone was not enough to establish

[22] Respondent herein also reacted directly to Dr. Steinman's non-medical exegesis into Vaccine Program legal standards, noting that the case he cited to defend his view that a known vaccine should be favored as causal over a non-specifically identified infection did not quite mean what he represented. Opp. at 27–28, *citing Torday v. Sec'y of Health & Hum. Servs.,* No. 07-372V, 2009 WL 5196163, at *3–4 (Fed. Cl. Spec. Mstr. Dec. 10, 2009). *Torday,* Respondent maintained, stood only for the proposition that when it was undisputed that the vaccine at issue *and* an unspecified illness could be causal, and the evidence was otherwise deemed close, it was reasonable to find the vaccine as causal. Here, by contrast, Dr. Collins and Respondent did *not* concede the Tdap vaccine was causal. Opp. at 28.

Respondent's reading of *Torday* is superior to Dr. Steinman's—and it only underscores why medical/scientific experts like Dr. Steinman are better off not opining on the meaning of Program caselaw or the applicable legal standards

molecular mimicry as a likely causal mechanism, since homology more often than not when occurring does not lead to autoimmune diseases. *Id.* at 29. Rather, more must be shown—but, Respondent noted, it could not (especially in the face of studies cited by Dr. Collins, like Baxter II, showing no association between the Tdap vaccine and GBS). *Id.*

The "did cause" prong was also unsatisfied, Respondent argued, given the evidence suggesting Petitioner's intercurrent infection was causal (coupled with an absence of record proof suggesting the presence of an aberrant immune response). Opp. at 30–31. Petitioner's timeframe arguments solely relied on Schonberger, which involved the flu vaccine (and as noted above would not be applicable in any event to the adjuvant causation theory). *Id.* at 31–32. And Respondent contended that Petitioner's evidentiary failings, as identified above, meant the burden had never shifted to Respondent to prove *any* alternative cause for Petitioner's GBS (despite the record evidence strongly suggesting there was in fact such an explanation, in the form of the unidentified infection). *Id.* at 32–33.

Reply

On reply, Petitioner reiterated his argument that his causal theories were preponderantly established (although he more clearly embraced molecular mimicry as his favored theory). Reply at 7 (specifically "putting aside the adjuvant theory" as "unnecessary" to succeed on the first prong). However, he also modified his legal argument a bit as far as the standard applied to the theory. He now argued that a somewhat-recent Federal Circuit decision (issued shortly before his first brief had been filed) had "clarified" (in connection with another intervening Court of Federal Claims determination)[23] that the standard for the first prong was only "biologically plausible" rather than a preponderance. Reply at 3 (*citing Kottenstette v. Sec'y of Health & Hum. Servs.,* 861 Fed. Appx. 433 (Fed. Cir. 2021)). And because molecular mimicry was an accepted and reliable theory for how autoimmune disease processes occur, it met that standard, given Dr. Steinman's detailed showing of homology as well as likely disease target antigens where a cross-reaction would be expected to occur. Reply at 7–8.

Petitioner noted again that there was an absence of clear proof of *what* the intercurrent infection was that Petitioner had experienced (and certainly limited direct proof that it was EBV

(although as discussed in this Decision, I give no weight at all to Dr. Steinman's legal pronouncements, and do not otherwise deem *Torday* a useful guiding decision).

[23] *J. v. Sec'y of Health & Hum. Servs.,* No. 16-864V, 2021 WL 3627107, at *19 (2021). In this single Court decision, one Court of Federal Claims judge proposed that the standard enunciated in another Federal Circuit decision, *Boatmon v. Sec'y of Health & Hum. Servs*., 941 F.3d 1351, 1359 (Fed. Cir. 2019), had been distinguished as not otherwise displacing a prior "plausibility" standard—despite the plain language of *Boatmon* to the contrary. Putting aside, however, that this determination in no way governs the outcome of this matter, I also find (as discussed below) that it does not provide helpful readings of Federal Circuit law on the question of the proper legal standard in Vaccine Act cases.

or CMV, despite suggestions to the contrary by Respondent), while the evidence of the vaccination was undisputed, making it an inherently superior explanation for Petitioner's GBS. Reply at 4–6. And regardless of other possible explanations, Petitioner claimed he had shown that the Tdap vaccine could be a substantial factor as well, entitling him to compensation. *Id.* at 10.

IV. Relevant Legal Standards

A. *Standards for Vaccine Claims*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[24] In this case, Petitioner cannot assert a Table claim based on CIDP.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health & Hum. Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a

[24] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury."

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras*, 121 Fed. Cl. at 245.

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) ("[h]owever, in the past we have made clear that simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof" (citing *Moberly*, 592 F.3d at 1322)). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored

in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review denied*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review denied* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Law Governing Analysis of Fact Evidence*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [ ] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained

in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs*., No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs*., 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.,* 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs*., 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not

be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs*., No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are

employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs*., No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review denied*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not every filed item factors into the outcome of this Decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.,* 527 F. Appx. 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

E. *Standards for Ruling on the Record*

I am resolving Petitioner's claim on the filed record. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The decision to rule on the record in lieu of hearing has been affirmed on appeal. *Kreizenbeck v. Sec'y of Health & Hum. Servs*., 945 F.3d 1362, 1366 (Fed. Cir. 2020); *see also Hooker v. Sec'y of Health & Hum. Servs*., No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided case on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec'y of Health & Hum. Servs*., 38 Fed. Cl. 397, 402–03 (1997) (determining that special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417; *Murphy v. Sec'y of Health & Hum. Servs.*, No. 90-882V, 1991 WL 71500, at *2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991).

ANALYSIS

I. Prior Reasoned Determinations Involving Tdap and GBS

There is a large body of reasoned decisions[25] discussing the alleged association between the Tdap vaccine and GBS—but it cannot be said that the Program has developed a consistent view as to what the science preponderantly "says" about the subject. Overall, it appears that the outcome in such cases is mostly a function of the evidence before the special master, with no clear trend one way or the other.

Thus, several cases decided in the past ten years found no causal association between Tdap vaccine and GBS. *See,* e.g., *Winkler v. Sec'y of Health & Hum. Servs.,* No. 18-203V, 2021 WL 6276203 (Fed. Cl. Spec. Mstr. Dec. 10, 2021), *mot. for review docketed,* Jan. 10, 2022 (ECF No. 62); *Montgomery v. Sec'y of Health & Hum. Servs.,* No. 15-1037V, 2019 WL 2511352 (Fed. Cl. Spec. Mstr. May 21, 2019); *Tompkins v. Sec'y of Health & Hum. Servs.,* No. 10-261V, 2013 WL 3498652 (Fed. Cl. Spec. Mstr. Jue 21, 2013), *mot. for review den'd,* 117 Fed. Cl. 713 (2014); *see also Isaac v. Sec'y of Health & Hum. Servs.,* 108 Fed Cl. 743 (2013) (affirming special master denial of claim alleging tetanus vaccine was causal of GBS), *mot. for review den'd,* 540 Fed. App'x 999 (Fed. Cir. 2013). These cases and the present matter have much in common.[26]

[25] As already noted, although prior decisions from different cases do not control the outcome herein, special masters may reasonably take into account, for guidance, the logic of reasoned entitlement determinations. In fact, it is wise to do so, given how often similar causation theories or fact patterns arise in Vaccine Program cases.

[26] I recently have denied entitlement in two cases where the petitioner alleged that a somewhat-related peripheral neuropathy featuring demyelination, CIDP, was caused by the Tdap vaccine. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 18-1012V, 2022 WL 1013264 (Fed. Cl. Spec. Mstr. Mar. 11, 2022); *Houston v. Sec'y of Health & Hum. Servs.,* No. 18-420V, 2021 WL 4259012 (Fed. Cl. Spec. Mstr. Aug. 19, 2021). However, CIDP is a distinct illness from GBS, despite their sharing of some features—and my holdings relied in part on finding that arguments regarding

In *Winkler,* for example, the petitioner (as here) offered molecular mimicry as his causal theory, noting also that certain known causes of GBS, like a *Campylobacter* infection, are understood to have molecular mimicry as their mechanism. *Winkler,* 2021 WL 6276203, at *23. But the special master determined that the claim turned not on the petitioner's first prong success, but rather on the fact that he had a demonstrated gastrointestinal infection, and that this infection was more likely causal (making it impossible to find that the vaccine "did cause" GBS). *Id.* at *23–25. Thus, evidence of a petitioner's intercurrent illness was dispositive, regardless of the persuasiveness of the causal theory.

Montgomery, by contrast, expressly found that a theory comparable to that offered herein was insufficient. There as here, the petitioner relied on Dr. Steinman, who opined (in what the special master deemed a "less-developed theory")[27] that the aluminum adjuvant in the Tdap vaccine triggered an aberrant autoimmune response. *Montgomery,* 2019 WL 2511352, at *5. But the special master rejected the theory, observing that critical details necessary to render the overall theory preponderant (such as the degree of cytokine upregulation stimulated by the adjuvant, or the duration of the response, which would implicate the initial, innate immune response) were missing. *Id.* Facially, it is difficult to ascertain how the showing made in the present case was any better.

Tompkins is an older decision, but its age underscores the extent to which (in the ensuing period) there have been few scientific discoveries or breakthroughs over the past several years that might better establish a Tdap-GBS association. The *Tompkins* special master denied entitlement in a case alleging that a number of vaccines received at the same time, including the Tdap vaccine, caused a petitioner's GBS, but the causal theory put forward attempted to assert that the vaccines could also *individually* trigger the disease. *Tompkins,* 2013 WL 3498652, at *15. The petitioner's expert, however, relied heavily on VAERS passive surveillance data,[28] and otherwise invoked a

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a GBS-Tdap association could not simply be superimposed on a case involving a distinguishable injury. *Sanchez,* 2022 WL 1013264, at *23; *Houston,* 2021 WL 4259012, at *17–18.

[27] The *Montgomery* petitioner seems to have primarily maintained that a human papillomavirus vaccine, administered at the same time as the Tdap vaccine, had caused her GBS. *Montgomery,* 2019 WL 2511352, at *4–5. That theory (which as here relied on molecular mimicry) was rejected as unreliable, despite the kind of BLAST search-homology showing also made in this case. *Id.*

[28] The Vaccine Adverse Event Reporting System ("VAERS") is a national warning system designed to detect safety problems in U.S.-licensed vaccines. *See About VAERS*, VAERS, https://vaers hhs.gov/about.html (last visited Apr. 5, 2022). It is managed by both the CDC and the FDA. VAERS monitors and analyzes reports of vaccine related injuries and side effects from both healthcare professionals and individuals. But it has been observed in the Program that VAERS data is not particularly probative of causation unless supplemented with other reliable evidence—since a VAERS report only establishes a temporal, post-vaccination occurrence, and thus shines no light of causation itself. *See also Vig v. Sec'y of Health & Human Servs.*, No. 01–198V, 2013 WL 6596683, at *17 (Fed. Cl. Spec. Mstr. Nov. 14, 2013) ("VAERS is a stocked pond, containing only reports of adverse events after vaccinations but no data about the number of vaccines administered or the occurrence of the same adverse event in individuals who have not been vaccinated").

number of theories (molecular mimicry, or endotoxin in tetanus-containing vaccines) that were only cursorily discussed. *Id.* at *19–23.

But there are also a few recent cases going the other way. *See,* e.g., *Mohamad v. Sec'y of Health & Hum. Servs.,* No. 16-1075V, 2022 WL 711604 (Fed. Cl. Spec. Mstr. Jan. 27, 2022). The *Mohamad* special master (who also decided *Montgomery*) ruled in the petitioner's favor in a Tdap-GBS case—but the analysis largely turned on the experts' disagreement as to whether (based on the demonstrated evolution of the government's published scientific/medical conclusions about Tdap causality) it could be said that the government had effectively *conceded* causation. The special master reviewed publications setting forth the government's view at different times, as well as some of the underlying studies it relied upon, concluding that in fact the Government had acknowledged that the vaccine *could* (albeit rarely) be causal, and therefore (primarily due to this admission) the first prong was met. *Mohamad,* 2022 WL 711604, at *17–18. This analysis included a brief discussion of the 2012 IOM Report filed by Petitioner in this case, as well as the Program's subsequent treatment of the issue along with two subsequent publications. *Id.* at *13–15. The special master in *Mohamad* gave special emphasis to a 2019 publication that (consistent with the package insert offered in this case) offered as a "precaution" that Tdap was to be carefully considered for individuals who previously had experienced GBS within six weeks of a tetanus toxoid-containing vaccine's prior receipt. *Id.* at *15.

A few important points distinguish *Mohamad*, however (besides the obvious fact that it does not control this outcome—and that almost all of the publications it evaluated were not offered as evidence herein). First, that special master appears to have been presented *only* with the argument that the Government had conceded the first prong, with both side's experts devoting most of their attention to that question (as opposed to the immunologic capacity of the vaccine to be causal—a matter that *has* been addressed a length in this case by Drs. Collins and Steinman). *Mohamad,* 2022 WL 711604, at *18 n.27. Second, there were specific issues with the credibility of Respondent's experts, and the resulting persuasiveness of their arguments. *Id.* at *8–9, *16–17. Also, the facts of this case do not present the circumstances for the precaution that the *Mohamad* special master deemed significant; it is not established on this record that Petitioner previously experienced GBS after receipt of a dose of a tetanus toxoid-containing vaccine. And there was far less obvious evidence of an intercurrent infection that could explain the petitioner's GBS than here—making the present case far more similar to *Winkler*.

Another recent case more supportive of a Tdap-GBS association (and cited specifically by Petitioner in his ruling on the record brief) is *Swaiss v. Sec'y of Health & Hum. Servs.,* No. 15-286V, 2019 WL 6520791 (Fed. Cl. Spec. Mstr. Nov. 4, 2019). But it too is distinguishable. There, a special master determined that small fiber neuropathy (characterized in *Swaiss* as "a variant" of GBS) could be caused by the Tdap vaccine via the mechanism of molecular mimicry. *Swaiss,* 2019 WL 6520791, at *23–27. But small fiber neuropathy is not the injury alleged in this case.

Moreover, the *Swaiss* special master acknowledged that the evidence offered to associate GBS and Tdap *generally* was somewhat lacking (although a favorable prong one determination was still made, based on the special master's reasoning that research into the causes of small fiber neuropathy was extremely limited, and that it would be therefore unfair to petitioner to hold the paucity of such evidence against him). *Id.* at *27. No such considerations obtain in this case, which involves the "classic" form of GBS, AIDP—a far more well-studied version.

It is true, as Petitioner's Reply observes, that reasoned determinations involving Tdap vaccine and GBS are dwarfed by the many instances in which prior Program cases alleging GBS after the Tdap vaccine have *settled*. Reply at 8–9, 9 n.6; *see also Mohamad,* 2022 WL 711604, at *19–20 (listing cases). But settlements cannot be invoked against the settling party to suggest a concession or waiver, and they do not otherwise provide reasoned guidance of any kind. *See,* e.g., *Randazzo v. Sec'y of Health & Hum. Servs*., No. 18-1513V, 2021 WL 829572, at *4 (Fed. Cl. Spec. Mstr. Feb. 1, 2021) (discussing low relevance of settled SIRVA claims in comparison to reasoned decisions). In such settled cases, no formal determination has been made as to the substance of the claim, and Respondent's determination to settle such claims cannot be construed fairly to reflect the tacit view that such claims have validity (since parties settle for many reasons beyond their assessment of a claim's actual merits). I thus do not factor these kinds of cases into my determination.

II. Petitioner Has Not Carried His Burden of Proof

A. *Petitioner Mischaracterizes his Prong One Burden*

Before discussing the success of Petitioner's *Althen* prong one showing, review of his framing of the legal standard applicable is warranted. For Petitioner *fully misstates* that standard, proposing a version that, if adopted, would obliterate the existing distinction between Table and non-Table claims in the Vaccine Program. Reply at 3.

As I noted above, the most recent controlling/precedential Federal Circuit caselaw directly addressing the subject states explicitly that the first *Althen* prong requires a preponderant showing—just like the other two prongs. *Boatmon*, 941 F.3d at 1359; *LaLonde,* 746 F.3d at 1339; *see also Moberly*, 592 F.3d at 1322. *LaLonde*, decided eight years ago, provides some insight into the reasoning for this standard. There, the Circuit was presented with a petitioner's appeal from a special master's determination that the DTaP vaccine (the version of Tdap administered to infants) had not caused a 17-month old minor to experience a focal brain injury. *LaLonde,* 746 F.3d at 1336–37. The special master deciding the case determined that the petitioner's expert had admitted in several regards that he could not substantiate his opinion with independent reliable evidence. *Id.* at 1338. On appeal, however, the petitioner referenced *Althen*'s oft-cited admonition that "the purpose of the Vaccine Act's preponderance of the evidence standard is to "allow the finding of

causation in a field bereft of complete and direct proof of how vaccines affect the human body" (*Althen,* 418 F.3d at 1280), relying on that to argue in turn that the special master had required proof of a precise biologic mechanism, contrary to the Circuit's determination in *Knudsen*. *Id.* at 1339. In effect, the petitioner argued, the special master was demanding scientific certainty.

The *LaLonde* majority disagreed. What the special master had done was *not* mandate that a specific mechanism be shown, but had instead "merely required that [the expert] support his testimony with a reputable or scientific explanation that pertained specifically" to the relevant facts. *LaLonde,* 746 F.3d at 1340. More importantly, in so doing the Circuit (referencing its earlier *Moberly* decision) emphasized that (consistent with the Act's express requirements) mere "identification" of a plausible theory did not meet the "statutory standard of preponderance of the evidence." *Id.* at 1339. In other words, the expert's recited theory lacked a sufficiently reliable foundation to establish causation—*independent* of whether the theory *on its face*, and when given an expert's imprimatur, was plausible.

Moberly, decided four years prior to *LaLonde*, reached a consistent result—but even more strongly demonstrates the rationale for requiring a preponderant showing on the first *Althen* prong. *Moberly* also involves a DPT-containing vaccine, and (consistent with *LaLonde*) an injury to an infant rather than adult. After entitlement was denied, the petitioners on appeal maintained that the special master had elevated their prong one burden, requiring scientific certainty rather than mere preponderance. *Moberly,* 592 F.3d at 1321. The Circuit cited the evidentiary standard set forth in *Althen*, and then observed as follows:

> While the petitioners acknowledge that the statute requires proof of causation by a preponderance of the evidence, see 42 U.S.C. § 300aa–13(a)(1)(A), they appear to be arguing for a more relaxed standard. They repeatedly characterize the test as whether [the infant's] condition was "likely caused" by the DPT vaccine. By that formulation, however, they appear to mean not proof of causation by the traditional "more likely than not" standard, but something closer to proof of a "plausible" or "possible" causal link between the vaccine and the injury, **which is not the statutory standard**. Similarly, the petitioners object to the use of the term "causation in fact" by the special master and the Court of Federal Claims, because they claim that proof that a vaccine "in fact" caused an injury would require conclusive scientific evidence. But **this court has regularly used that term to describe the causal requirement for off-Table injuries and has made clear that the applicable level of proof is not certainty, but the traditional tort standard of "preponderant evidence."**

Id. at 1322 (emphasis added). The *Moberly* panel went on to note that although causation was presumed in Table cases (which have other more lenient features favorable to claimants), "the tort standard of causation is applicable" to non-Table claims. *Id.* As a result, to argue for a lesser

standard for causation in the non-Table context was to "conflate the burden of proof imposed for off-Table injuries with the lenient presumptions applicable to Table injuries." *Id.*

Nothing the Circuit has decided in the intervening ten to fifteen years suggests that this preponderant requirement was a temporary frolic and detour, pivoting momentarily from the "norm" of plausibility. It was embraced as recently as 2019. *Boatmon,* 941 F.3d at 1360 ("[w]e have consistently rejected theories that the vaccine only "likely caused" the injury and reiterated that a "plausible" or "possible" causal theory does not satisfy the standard"). I note also that requiring preponderance for each individual *Althen* prong arises from a reasonable reading of not only the Act but the *Althen* decision itself. The Act specifically states that petitioners must demonstrate "by a preponderance of the evidence the *matters* required by the petition." Section 13(a)(1)(A) (emphasis added). It does not say that some "matters" need not be preponderantly established. And in *Althen's* syntax, preponderance stands *outside*, and hence modifies, all *three* of its enumerated prongs. *Althen*, 418 F.3d at 1278.[29] There is no logical reason to read it as requiring preponderance when establishing the vaccine "did cause" injury, but not when a petitioner must demonstrate his satisfaction of the "can cause" element.

Petitioner incorrectly maintains that the Circuit in *Kottenstette* altered the above understanding. Reply at 3. *Kottenstette* was yet another case involving the DTaP vaccine allegedly causing an infant injury, and it does comment on *Boatmon—but not to make the point that Boatmon misstated the evidentiary standard applied to Althen prong one*. Rather, the *Kottenstette* panel was *distinguishing* the *Boatmon* panel's determination (in affirming reversal of the special master's entitlement determination) that the deciding special master in that case had applied the wrong burden of proof in assessing the claimant's showing. *Boatmon*, 941 F.3d at 1359 ("[t]he Special Master deviated from the correct 'reputable,' 'sound and reliable' standard and articulated a lower 'reasonable' standard" in considering the first *Althen* prong). The *Kottenstette* panel, by contrast, found that the special master involved in the decision it was reviewing[30] had articulated the *proper* overall preponderant requirement—and thus (in the *Kottenstette* panel's view) had not committed the same error of mistakenly construing the burden as in *Boatmon*. *Kottenstette,* 861 F. App'x at 440–41. The *Kottenstette* panel thus does not characterize the *Boatmon* articulation of the first

[29] "Concisely stated, Althen's burden is to show *by preponderant evidence* that the vaccination brought about her injury *by providing*: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Althen* 418 F.3d at 1278 (emphasis added).

[30] The procedural history in *Kottenstette* was somewhat complicated. There, a special master after hearing decided a claim in the petitioners' favor, and not long after retired. On appeal, the Court reversed, and the matter was remanded to a different special master, who re-decided the case in light of the legal standard articulated by that judge. *Kottenstette,* at 434–35. The appeal to the Circuit was based on the revised finding, which was now unfavorable to the petitioners, and the Circuit's analysis took into account how the *original* special master presiding over the case had applied the law initially (in favor of entitlement).

Althen prong standard to be erroneous, nor does it say anywhere that mere plausibility is sufficient to establish the first *Althen* prong.

Petitioner's reading of *Kottenstette* is thus wholly inconsistent with the principles differentiating Table claims and causation-in-fact claims—a distinction observed in *Moberly*. The significance of this distinction would be utterly lost if Petitioner's plausibility standard governed the "can cause" prong. For a standard of mere plausibility applied to the first Althen prong would be *one that claimants would never fail to meet, and would thus be functionally equivalent to a Table claim's causation presumption*. The general intuition that a vaccine preceding an injury ***might*** have caused that injury is easily fleshed out to the level of plausibility with some scientific evaluation of a vaccine's components, and how they could, in theory, chemically cause an aberrant immune response. There is no shortage of experts who would be willing to so opine; indeed, even Respondent's experts consistently admit at trial, in my experience, that it is not "impossible" for a given vaccine to cause an injury in a susceptible person, given how little remains known about the precise functioning of the immune system. But the Vaccine Act was not intended to so lower a petitioner's burden for causation-in-fact claims—even if scientific certainty is never the proper standard.

B. *Petitioner Has Not Preponderantly Met his Burden of Proof*

1. *Althen* Prong One

Two causation theories were ultimately advanced in this case, but neither was preponderantly established.

Alum as Adjuvant Theory

It was wise of Petitioner to "supplement" his case with a second causation theory, for the first one Dr. Steinman proposed—that alum included in the Tdap vaccine as an adjuvant can produce demyelination—was woefully inadequate.[31]

Dr. Steinman's theory heavily relies on what is known about the *intended* function of the aluminum adjuvant—and certainly the substantiating support offered for these small contentions was reliable. It was also reasonably submitted that cytokines play *some* role in GBS pathogenesis, at various points in the disease's course. But as I have observed in numerous prior cases, claimants cannot prevail simply by pointing to how vaccines are expected to function, and then extrapolating such contentions into the basis for an argument about pathologic process that is not *also* corroborated with vaccination. *Palattao v. Sec'y of Health & Hum. Servs.,* No. 13-591V, 2019 WL 989380, at *36 (Fed. Cl. Spec. Mstr. Feb. 4, 2019) ("claimants cannot transmute scientific evidence

[31] Indeed, Petitioner largely seems to have walked away from his first theory by the time of his reply brief.

exploring how vaccines normally function in the immune system into a reliable and persuasive causation theory that *any* vaccine can be pathogenic without a more specific showing that applies to the circumstances at hand,") (*citing Olson v. Sec'y of Health & Hum. Servs.,* No. 13-439V, 2017 WL 3624085 (Fed. Cl. Spec. Mstr. July 14, 2017), *mot. for review den'd*, 135 Fed. Cl. 670 (2017), *aff'd*, 2018 WL 6721291 (Fed. Cir. 2018)).

Thus, these individual components of the theory, as reliably established as they were, were not persuasively connected to *other* preponderant evidence to establish that the Tdap vaccine *likely* can cause GBS solely on the basis of the inclusion of the aluminum adjuvant. Petitioner could point to no direct proof on the subject—and while he was not *required* to do so, the absence of evidence (as reliably shown by Dr. Collins) that the inclusion of aluminum as an adjuvant is the "X factor" leading to GBS meant that Petitioner needed much more other additional circumstantial proof to connect all the dots. Dr. Collins also successfully demonstrated that direct studies pertaining to adjuvanted vaccines did not produce outcomes consistent with Petitioner's theory. *See,* e.g., First Collins Rep. at 7. For if it is the adjuvant that is causal, then *any* vaccine containing it should be associated with an increased risk of GBS—but this is not the case.

I have before rejected this kind of adjuvant-centered theory in other contexts.[32] *Zumwalt v. Sec'y of Health & Hum. Servs*., No. 16-994V, 2019 WL 1953739, at *18 (Fed. Cl. Spec. Mstr. Mar. 21, 2019), *mot. for review den'd,* 146 Fed. Cl. 525 (2019) ("[t]he fact that vaccines are known to stimulate cytokine production (in part due in some cases to the inclusion of an adjuvant) does not amount to a reliable causation theory that such stimulation is necessarily disease-causing"). Nothing about how the theory was presented in this case made it more likely. And the Tdap package insert (which at most provides indirect support for a vaccine association, in circumstances distinguishable from the present) is weak proof supporting causation as a general matter. *Werderitsh v. Sec'y of Health & Human Servs.,* No. 99–319V, 2005 WL 3320041, at *8 (Fed. Cl. Spec. Mstr. Nov. 10, 2005). At bottom, the adjuvant theory offered in this case over-relies on a conclusion that a component common to many vaccines, and included for the specific purpose of heightening immunogenicity, can promulgate disease—in the face of ample evidence that it "more likely than not" does not do so.[33]

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[32] For example, petitioners have argued that aluminum is a toxic metal that harms the body—despite the fact that individuals *naturally* ingest greater amounts of aluminum than via vaccination, but without attendant harm. *McKown v. Sec'y of Health & Hum. Servs.*, No. 15-1451V, 2019 WL 4072113, at *34 (Fed. Cl. Spec. Mstr. July15, 2019). In other cases, claimants' experts have proposed a theory entitled "autoimmune/inflammatory syndrome induced by adjuvants," or "ASIA"—but special masters have noted that it lacks both medical community acceptance as well as overall substantiation. *Pearson v Sec'y of Health & Hum. Servs.*, No. 17-489V, 2019 WL 1150044, at *11 (Fed. Cl. Spec. Mstr. Feb. 7, 2019); *Garner v. Sec'y of Health & Hum. Servs.*, No. 15-063V, 2017 WL 1713184 (Fed Cl. Spec. Mstr. Mar. 24, 2017), *mot. for review den'd*, 133 Fed. Cl. 140 (July 31, 2017). Admittedly, Dr. Steinman's theory is somewhat distinguishable—but was no more credible.

[33] By contrast, other vaccines have been credibly associated with GBS *without consideration of the presence of an adjuvant at all*. Thus, the theory that the flu vaccine can cause GBS is not only far better corroborated (so much so that it constitutes a Table claim—and hence the Government recognizes enough science supports it for causation to

Dr. Steinman objects to some of Dr. Collins's arguments that individual items of literature he has offered were "cherry picked," and do not by themselves prove his theory, maintaining in defense of their reference that they are meant to be viewed collectively. This objection has some merit. First, it is unquestionably the case (as the Circuit captured in the *Althen* opinion, with its phrase "a field bereft of complete and direct proof of how vaccines affect the human body" (*Althen*, 418 F.3d at 1280)) that Program petitioners can *never* be faulted for their inability to brandish a single, all-encompassing and scientifically-reliable piece of evidence showing that a vaccine is clearly causal of an injury. That kind of evidence simply does not exist.[34] Second, the very fact that the Program combines a preponderant, "more likely than not" causation standard with very few (if any) limits on the kinds of evidence that can be offered to meet it, means that claimants may satisfy their burden by connecting individual items of proof (circumstantial or otherwise) that, individually, only partially address the greater issue.

But *this does not mean* that tying together items standing for reasonable and reliable, but very narrow, points will always produce a theory that is *in total* preponderantly established. The causal chain an expert forges must do more than simply make leaps from "stone to stone" without some additional support—and without proposing a reliable reason for making the leap. Thus, the argument that (a) vaccines cause cytokine upregulation, (b) cytokines are part of the pathogenic process in GBS, therefore (c) vaccines can cause GBS, leaves out a number of necessary intermediate steps, such as (a) *when* can the amount of cytokine production generally become excessive, (b) *does* this occur with vaccination, and (c) *how* central are those cytokines to the relevant disease process (and in the case of vaccination, they must be an *initial* instigating factor). The absence, or presence, of other corroborative evidence (e.g., what environmental factors are deemed causal of the injury that are comparable to a vaccine, or is anything scientifically known about the disease's biologic mechanisms) can also be significant. Here, that necessary connective evidence was wholly lacking, and I therefore cannot conclude that the causation theory relying on the alum adjuvant was preponderantly established.

Molecular Mimicry Theory

Petitioner's second theory was one that, more often than not in Program cases, is the *primary* theory offered to explain how virtually any vaccine could produce an autoimmune disease—via molecular mimicry. But not only was it proposed late in the game, but it too was not preponderantly shown to be likely causal. All Dr. Steinman did was offer evidence suggesting a

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be conceded), but does *not* at all rely on alum as an adjuvant, since the flu vaccine most often administered does not contain one.

[34] Indeed, even the flu vaccine-GBS association is mainly based on a string of propositions, including (a) that molecular mimicry between flu vaccine components and nerve cross-attack targets can be shown, and (b) that an epidemiologic study now over 40 years old reliably established a high incidence of GBS after receipt of the flu vaccine, when compared to those who did not receive it.

theoretic *possibility* of homology between Tdap vaccine components and locations in the nerve structures where an autoimmune attack might occur. But this is a relatively easy showing to make, given the prevalence of homology in nature. It does *not* amount to a showing that a cross-reaction instigated by the Tdap vaccine resulting in GBS is "more likely than not." And Dr. Collins offered evidence and testimony raising reasonable questions about whether every variant of GBS inherently unfolds *only* from molecular mimicry causing the production of cross-reactive autoantibodies, or only at the identified myelin targets. *See,* e.g., Fourth Collins Rep. at 2-5; Orlikowski.

I once again emphasize (as I have in many prior cases) that molecular mimicry is not a "get out of jail free card" in the Program, entitling claimants who hire Dr. Steinman (or someone else sufficiently conversant with molecular biology and the relevant databases) to compensation, merely because it has scientific reliability as a general matter. *Yalacki v. Sec'y of Health & Hum. Servs*., No. 14-278V, 2019 WL 1061429, at *34 (Fed. Cl. Spec. Mstr. Jan. 31, 2019), *mot. for review den'd,* 146 Fed. Cl. 80 (2019) ("it is not enough for a claimant to invoke the concept of molecular mimicry along with *some* identified homology between an amino acid sequence and a target antigen in order to carry her burden"). Without some reason to further find that the relevant vaccine *can* be causal of the specific injury at issue—however that might be demonstrated—establishing a potentiality for molecular mimicry alone does not meet the preponderant standard of proof, no matter what degree of amino acid identity (sequential or not) Dr. Steinman can demonstrate with a BLAST search.

Epidemiologic evidence offered by Respondent also undercuts Petitioner's showing.[35] Baxter II—a large-scale study identifying no association between Tdap and GBS—was particularly harmful. It was far more relevant to Petitioner's claim herein than Baxter I (authored by almost all of the same individuals as Baxter II), which Dr. Steinman favorably cited but which only showed a possible association between Tdap and *ADEM*—a distinguishable central nervous system-impacting demyelinating disease. The very fact that Dr. Steinman chose to cite Baxter I is telling—if one epidemiologic study not fully on point is nevertheless supportive of his theory, how can a study *directly* on point (since it involved the injury at issue *in this case*), and written by largely the same group of scientific professionals, not *also* bear on the case's outcome?

[35] Even though Program petitioners are never *obligated* to offer epidemiologic evidence, it is well-established by controlling Federal Circuit caselaw that relevant epidemiologic evidence *can be taken into account* by a special master in weighing a claimant's success on the first *Althen* prong. *Taylor v. Sec'y of Health & Human Servs.*, 108 Fed. Cl. 807, 819–21 (Fed. Cl. 2013) (special master did not err in considering epidemiological evidence); *Andreu*, 569 F.3d at 1379 (a special master may assess epidemiological evidence in "reaching an informed judgment as to whether a particular vaccination likely caused a particular injury.").

Such evidence is reasonably subject to the same reliability and methodology considerations that apply to all scientific/medical evidence offered, and it should not be viewed as dispositive *per se.* But it deserves weight when it exists, and it can serve to weaken confidence in a claimant's causal showing.

My evaluation of Dr. Steinman's theory also takes into account, to a small extent, conduct reflected in his reports that undermined his persuasiveness. This case provides yet another example of Dr. Steinman's unfortunate proclivities as an expert when offering opinions in Program cases. Vaccine Program experts should confine their testimony to the *scientific* and *medical* issues that their training and expertise renders them competent to comment upon. They should not discuss the legal standards that govern entitlement decisions (beyond perhaps offering testimonial caveats consistent with those standards).[36] Yet Dr. Steinman wasted numerous pages of his report (frequently appending the relatively-useless proviso that he is "not a lawyer") to *opining* directly on the proper application of those legal standards, and even invoked prior decisions as precedential support. *See,* e.g., Second Steinman Rep.at 1; Third Steinman Rep. at 1. I have in the past criticized Dr. Steinman for engaging in similar conduct. *See,* e.g., *Rolshoven v. Sec'y of Health & Hum. Servs*., No. 14-439V, 2018 WL 1124737, at *21 (Fed. Cl. Spec. Mstr. Jan. 11, 2018). He will continue to do harm to his credibility as an expert if he does not stop acting in this fashion.[37]

I emphasize that my reaction to the above is not the primary basis for my determination that the first *Althen* prong was not met. Rather, neither of the causation theories offered was preponderantly established with sufficient reliable medical or scientific evidence, despite Dr. Steinman's ample credentials to opine on the subject. But it is well within the bounds of the exercise of my duties as a special master to take into account the totality of facts pertaining to an expert's presentation in evaluating credibility and persuasiveness. The special master is not a "potted plant" in Vaccine Act proceedings, obligated to ignore what experts actually *say or do* in a case out of the concern that a negative reaction might prejudice the petitioner. Here—and in addition to the fact that the causal theories offered were based on unreliable science or medicine, and otherwise not preponderantly established—Dr. Steinman undermined his showing.

2. *Althen* Prong Two

[36] Thus, counsel often conclude their direct examination of an expert by asking the expert whether he or she holds the opinion offered "to a reasonable degree of medical probability," or whether something contended is simply "more likely than not."

[37] In addition, I note that the molecular mimicry theory was only offered as a substitute for an initial theory that over time proved problematic—and after multiple reports on that theory had been filed. I acknowledge that Dr. Steinman *purports* that the two theories are equally valid, and could *both* explain causation, alone or taken together. Fourth Steinman Rep. at 1. But (in my experience with Dr. Steinman as an expert—having seen his reports, or listened to his testimony, many times over the past eight years) molecular mimicry is more often than not the primary, if not sole, causation theory he espouses—and he did not embrace it in this case at the outset. Indeed, Dr. Steinman was comfortable relying on the adjuvant theory enough to defend it multiple times against Dr. Collins's attacks, and over a nearly three-year period, without any hedging or qualification. If this theory were so ironclad, *why* was there any need to supplement it with an alternative? While I have evaluated molecular mimicry on its own terms, and based almost wholly on the evidence and testimony offered for it, the fact that it was Petitioner's "second choice" suggests to a small degree a lack of faith in its applicability.

Several items of record evidence weigh against Petitioner's assertion that the Tdap vaccine he received likely "did cause" his GBS. In particular, the majority of immediate treaters did not propose vaccination to be causal, finding far more significant the indisputable evidence of an intercurrent URI. *See,* e.g., Ex. 3 at 15, 19–20 (Dr. Han), 36–37 (Dr. Deangelis), 41 (Dr. Given), 126–27 (Dr. Glodan). On the other hand, during an infectious disease consult Dr. Klirsfeld noted as well that the injury could be "possibly" vaccine-associated. Ex. 3 at 53. But this initial speculation never developed into an acting diagnostic hypothesis, and so the weight of treater views evidenced in this record does not support vaccine causation. (A subsequent dietician's mention of the vaccination deserves even less weight—not only is such a professional's view less informed than the neurologic or other disease specialist treaters who saw Dr. K.A., but the record at issue suggests this was simply mentioned as part of the patient history). Ex. 9 at 1064.

At the same time, the record reveals several instances in which treaters proposed that Petitioner's URI *did* play a role in his subsequent neurologic injury. I also do not give substantial, if any, weight to Dr. Steinman's objections that the term of art "influenza-like illness" to describe Petitioner's URI is scientifically indeterminate—since filed record evidence from competent medical and scientific professionals employ the term in their own studies. *See,* e.g., Ex. 3 at 47 (using the term "flu-like").

Dr. Steinman nevertheless objects to giving this evidence weight in the absence of proof of the specific nature of the infection. In so arguing, Petitioner notes correctly that the record is ambiguous and inconclusive on this point. Thus, initial testing for WNV was positive for a resolved, but not current, infection (Ex. 3 at 15–20, 53, 63), and Petitioner did not test positive for EBV (although evidence of a resolved CMV was identified). *Id.* at 268–85; Ex. 9 at 178–80. I cannot say on this record that the evidence preponderantly establishes the specific infectious agent behind Petitioner's URI.

But (putting aside the fact that Dr. Steinman relies in part on his own reading of Program case law for these contentions about infection specificity—despite his lack of expertise to so opine), it is incorrect that evidence regarding possible alternative causes should not be included in a special master's weighing, absent definitive proof of the nature of the infection. Section 13(b)(1)(A) (special master empowered to consider "any diagnosis, conclusion, medical judgment . . . which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness"). Here, not only is there incontrovertible evidence that Petitioner *first* experienced URI symptoms before neurologic-related symptoms, but also that the majority of his treaters deemed the URI most likely causal. The vaccine does not deserve greater weight simply because it is "known" whereas the precise nature of the infection is not. Indeed—medical science cannot always test for or identify a specific infection, as has been observed in prior cases. *Randolph v. Sec'y of Health & Hum. Servs.*, No. 15-146V, 2021 WL 5816271, at *21 (Fed. Cl. Spec. Mstr. Nov. 12, 2021) ("[c]ausation claims do not succeed merely because Respondent cannot prove with

certainty what *was* causal"). Dr. Collins also persuasively showed that many facially-reliable studies had included "influenza-like illness" as a potentially causal agent of GBS, despite Dr. Steinman's protestations. *See,* e.g., Tam; Stowe. And since scientific certainty is not the relevant evidentiary standard, it cannot be the case that Respondent must meet that standard in rebutting a petitioner's showing.

This is also not an instance in which what is referred to as a *Shyface* analysis assists Petitioner. There are circumstances where experts on both sides concede two or more factors could be causal of injury (including vaccination), resulting in entitlement for the petitioner if the special master concludes that the vaccine was at least a "substantial" factor (even if not the primary or predominant factor). *Deribeaux v. Sec'y of Health & Hum. Servs.*, 105 Fed. Cl. 583, 589 (June 4, 2012). But here, there was no concession by Dr. Collins that the Tdap vaccine *can* be causal at all[38]—and I have found that Petitioner did not preponderantly establish this to the case. Thus, the mere fact that temporarily Petitioner received the Tdap vaccine before onset of his URI does not compel me into a *Shyface* determination that the URI was not likely *solely* causal.

Overall, the medical records strongly support the conclusion that Petitioner's intercurrent URI likely caused his GBS—not that it was caused by the Tdap vaccine.[39]

3. *Althen* Prong Three

Petitioner's success in establishing that the timeframe for his onset of symptoms post-vaccination was medically acceptable (in terms of vaccine causation) is not consistent from theory to theory. Little was offered to substantiate the timeframe for Mr. K.A.'s 18 to 19-day onset (August 31, 2013, or September 1, 2013) under the theory that the adjuvant caused his GBS. Indeed, since this theory depended on an innate, cytokine-driven reaction to the vaccine, the

[38] As Respondent observed in his opposition brief, Dr. Steinman's misplaced reliance on *Torday* as requiring specification of a precise infection to counter the known quantity of a vaccination really invokes a *Shyface*-like analysis from a case where more than one factor was deemed potentially causal, leading the special master to give less weight to the unknown precise nature of the infection. *Torday,* 2009 WL 5196163, at *3–4.

[39] As noted, I do not find that Petitioner has established that the Tdap vaccine can cause GBS, so the burden of proof never shifted to Respondent to prove a "factor unrelated." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1354 (Fed. Cir. 2008). I have otherwise reasonably included, in my weighing of evidence for and against Petitioner, the record proof pertaining to Petitioner's URI, finding that it undercuts Petitioner's contentions that the Tdap vaccine was causal. *Stone v. Sec'y of Health & Human Servs.,* 676 F.3d 1373, 1380 (Fed. Cir. 2012) ("no evidence should be embargoed from the special master's consideration simply because it is also relevant to another inquiry under the statute"); *see also de Bazan,* 539 F.3d at 1353 ("[t]he government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the petitioner's evidence on a requisite element of the petitioner's case-in-chief").

But I also note that even if I had found that Petitioner had met his initial causation burden, the record before me would preponderantly establish that the URI was *more likely causal*—and hence Respondent on this record would have met his burden had it shifted. The record persuasively establishes that not only are a variety of infectious processes (including "influenza-like illness") associated with GBS more convincingly than the Tdap vaccine, but also that treaters overall proposed that Petitioner's own history was consistent with that relationship.

pathologic process would have likely occurred far closer in time to vaccination—suggesting that even an onset around three weeks post-vaccination would be too long. Opp. at 23, *citing Montgomery*, 2019 WL 2511352, at *5.

As noted, however, Petitioner seems to have opted to place more emphasis on Dr. Steinman's second causation theory—that molecular mimicry between vaccine antigens and self structures associated with the initial attack on the nerves was the mechanism driving the autoimmune process. Facially, a three-week onset for such a process is more medically reasonable, taking into account (a) how long the adaptive immune response (in which antibodies would be produced in reaction to vaccine antigens and then start cross-reacting with self tissues) would take generally, plus (b) literature like Schonberger. *Keja v. Sec'y of Health & Hum. Servs.*, No. 17-1511V, 2021 WL1736816, at *21 (Fed. Cl. Spec. Mstr. Apr. 2, 2021). Of course, Schonberger is not specific to Tdap vaccine at all, diminishing its evidentiary value. But *had I found* that the Tdap vaccine "can cause" GBS, then the fact that the timeframe of onset occurred when, from a medical standpoint, it would be expected to occur would inure to Petitioner's benefit. As it stands, however, and because the claim fails on the first two *Althen* prongs, Petitioner's ability to preponderantly support the third prong does not avail him.

III. This Case Was Appropriately Decided on the Papers

In ruling on the record, I am choosing not to hold a hearing—a determination that the parties largely accepted.[40] Determining how best to resolve a case is a matter that lies generally within my discretion, but I shall explain why I determined that a hearing was unnecessary.

Prior decisions have recognized that a special master's discretion in deciding whether to conduct an evidentiary hearing "is tempered by Vaccine Rule 3(b)," or the duty to "afford[] each party a full and fair opportunity to present its case." *Hovey*, 38 Fed. Cl. at 400–01 (citing Rule 3(b)). But that rule also includes the obligation of creation of a record "sufficient to allow review of the special master's decision." *Id*. Thus, the fact that a claim is legitimately disputed, such that the special master must exercise his intellectual faculties in order to decide a matter, is not itself grounds for a trial (for if it were, trials would be required in every disputed case). Special masters are expressly empowered to resolve fact disputes without a hearing—although they should only so act if a party has been given the proper "full and fair" chance to prove their claim.

[40] Petitioner's initial ruling on the record brief did not oppose deciding this case on the papers. On Reply, however, Petitioner included a footnote setting forth some process objections: that he was denied the opportunity to offer his own infectious disease expert (by the special master previously assigned to the case) to counter Dr. Collins's arguments, and that, although he noted my discretion to choose how best to decide the case, a hearing was warranted since the petitioner's theories were "closely contested." Reply at 5 n.4. Putting aside the underwhelming and somewhat eleventh-hour nature of this objection, I nevertheless determine (as explained herein) that the claim could be, and was, fairly adjudicated solely on the basis of the papers—and as already noted hearings are not held simply because the parties disagree on entitlement.

It was wholly fair to both sides to resolve this case on the papers and after briefing by the parties. Over the case's nearly six years, Petitioner was afforded the opportunity to offer *five* written expert reports—so many that the reports began to repeat prior arguments and/or devolve into expert bickering as they piled up. Petitioner was also permitted to modify his causation theory entirely, after Respondent voiced objections to its sufficiency. And the second theory proposed was one with which I have substantial familiarity—meaning I did not need to hear live testimony from Dr. Steinman to understand or react to it. It cannot be denied, given the overall procedural history, that Petitioner received a reasonable chance to prove his claim.

CONCLUSION

A Program entitlement award is only appropriate for claims supported by preponderant evidence. Here, Petitioner has not made such as showing. Petitioner is therefore not entitled to compensation.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[41]

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

[41] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.